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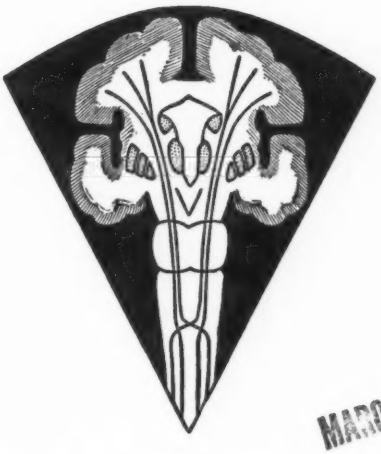

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1. Mathews et al.: Am. Pract. & Dig. Treat. 6:360 (Mar.) 1955.
2. Albert and Coakley: Anesth. & Analg. 33:285 (July-Aug.) 1955.

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The first investigation of a hemostat with an action comparable to Adrenosem Salicylate was made by Derouaux and Roskam[1] in 1937. They reported that an oxidation product of adrenalin, adrenochrome (which has no sympathomimetic properties), has prompt hemostatic activity.

It was further found that various combinations of adrenochrome, notably the oxime and semicarbazone, produced stable solutions. But, these were so slightly soluble that sufficient concentration could not be obtained for practical therapeutic use. By combining these adrenochrome compounds in a sodium salicylate complex a stable, soluble form can be obtained. This complex has been given the generic name, carbazochrome salicylate, and is supplied under the trade name Adrenosem Salicylate.

Roskam, in his study entitled "The Arrest of Bleeding,"[2] enumerates "the drugs whose efficaciousness as hemostatics have been proved by accurate methods in experimental animals and in healthy men as well. . . . One is the monosemicarbazone of adrenochrome [Adrenosem Salicylate]."

Chemistry

Adrenosem Salicylate is a synthetic chemical. The full chemical name is adrenochrome monosemicarbazone sodium salicylate complex.

Pharmacology

Although it is chemically related to epinephrine, Adrenosem Salicylate has no sympathomimetic effects. It does not alter blood components, nor does it affect blood pressure or cardiac rate.[2-7]

Sherber, in an early study,[3] concludes that Adrenosem Salicylate* "is a potent antihemorrhagic factor in those conditions in which the integrity of the smaller vessels is interrupted, and is superior to any similar material that is now available."

He continues, "From our experience it appears that adrenochromazone complex is indicated in preventing vascular accidents incident to hypertension; in maintaining small vessel integrity; in the preoperative preparation where oozing from a vascular bed is anticipated, as in tonsillectomies, adenoidectomies and prostatectomies; and as an adjunct in the treatment of bleeding from such surgical procedures."

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Clinical investigators[2-7] are in agreement that Adrenosem Salicylate controls bleeding and oozing by decreasing capillary permeability and by promoting the retraction of severed capillary ends. It aids in maintaining normal capillary integrity by direct action on the intercellular "cement" in capillary walls. The interesting work of Fulton[8] confirms this. Adrenosem Salicylate, since it is not a vasoconstrictor, has no effect on large severed blood vessels and arterioles.

Adrenosem Salicylate is being used both prophylactically and therapeutically in thousands of hospitals, and in virtually every type of surgical procedure. It has also proved most useful in dental surgery.[7]

Owings reported on the use of Adrenosem Salicylate in controlling postoperative adenoid bleeding in 102 cases.[4] "We have used 2½ mg.

(* U.S. Patent 2,581,850)

(½ ampule) intramuscularly, 15 minutes before anesthesia for children and 5 mg. (1 ampule) for adults." In only one patient did bleeding occur. Three others showed red blood from the nose and mouth. These patients "were then given 5 mg. intramuscularly, with prompt and complete control. We have also noticed that bleeding stopped more promptly on the operating table."

This is a 1% incidence of postoperative bleeding using Adrenosem Salicylate preoperatively, compared to an incidence of 10% postoperative bleeding in all cases taken from previous records, without Adrenosem Salicylate medication.

Peele reports on the use of Adrenosem Salicylate in treating 178 patients with 24 different conditions.[5] The drug was first used to control postoperative hemorrhage from the adenoid region. He adds: "The results were so dramatic that since that date [1953] Adrenosem Salicylate has been used postoperatively to reduce bleeding from all otolaryngologic and bronchoesophagolic procedures, to treat postoperative hemorrhage from the tonsil and adenoid regions, and to treat selected cases of epistaxis."

The effectiveness of Adrenosem Salicylate in controlling bleeding and oozing in 330 patients is reviewed by Bacala.[6] "Our experience of the effect of carbazochrome salicylate on 317 surgical indications and 13 obstetricogynecological conditions, has been therapeutically encouraging and successful for the control of capillary bleeding. Foremost among the cases studied were 223 tonsillectomies definitely benefited by this metabolic hemostat, making a diminution of the control incidence of posttonsillectomy bleeding of 19.8% down to 7%. It has also been found useful in gastro-intestinal bleeding, cataract extraction, epistaxis, incisional seepage, trans-urethral prostatectomy, menometrorrhagias, cervical ooze, antepartum and postpartum bleeding, threatened abortion, and prevention of capillary hemorrhages during hedulin or dicumerol therapy."

Side Effects

All investigators concur that, at recommended dosage levels, Adrenosem Salicylate is free from toxic effects. No cumulative effects attributable to the drug have been reported.

The only side reaction noted has been a transient stinging sensation in the area of injection when Adrenosem Salicylate is used intramuscularly. As one investigator comments: "The brief discomfort which attends the injection of Adrenosem into the gluteal region has not been a significant problem in children or adults as originally anticipated."[5]

Indications

Idiopathic purpura, retinal hemorrhage, familial telangiectasia, epistaxis, hemoptysis, hematuria.

Postoperative bleeding associated with:
tonsillectomy, adenoidectomy and
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uterine bleeding;
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Dosage

For recommended dosage schedules, please send for detailed literature.

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Tablets: 2.5 mg. S.C. Yellow, bottles of 50.
Syrup: 2.5 mg. per 5 cc. (1 tsp.), 4 ounce bottles.

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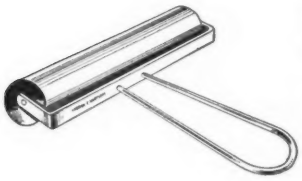

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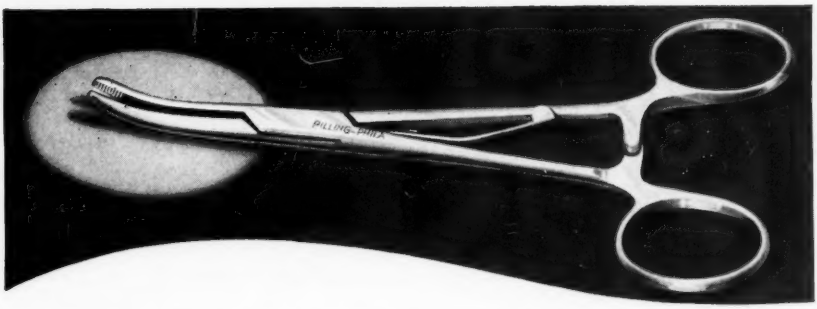

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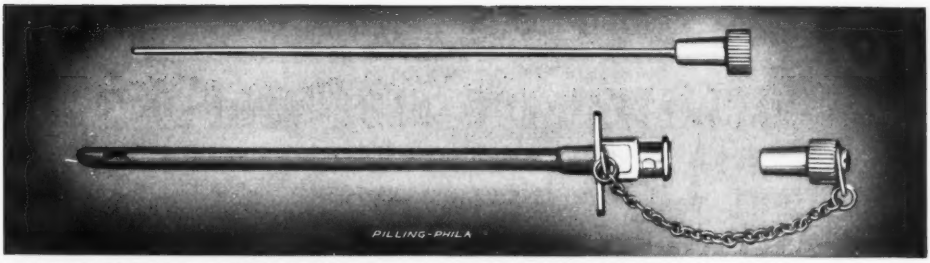

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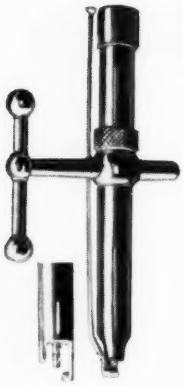

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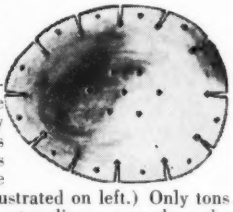

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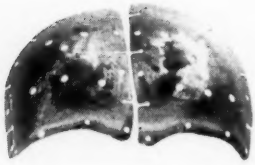

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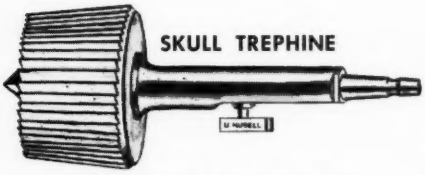

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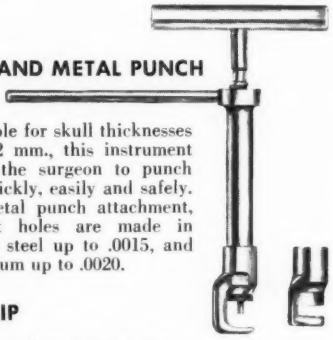

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THE ROLE OF ATLANTOID COMPRESSION IN THE ETIOLOGY OF INTERNAL CAROTID THROMBOSIS*

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(Received for publication September 30, 1955)

THROMBOSIS of the internal carotid artery, long recognized as a clinical and pathologic entity, is the subject of currently increased interest principally because, by means of cerebral angiography, its presence can be demonstrated during life and the diagnosis thereby established less equivocally. Since our knowledge of the prevention and treatment of this lesion and its sequelae leaves much to be desired, the continuing accumulation of clinical data and the study of potential etiologic factors seem justifiable.

In this paper, we present a clinical review of 24 cases of angiographically demonstrated occlusion of the internal carotid artery, complete or probably complete in all but one, and assumed in all to be caused by thrombosis. Our principal purpose, however, is to discuss the potential role of compression of the internal carotid in the neck by the lateral process of the atlas as an etiologic factor in some instances of this disease.

CLINICAL REVIEW

The major clinical aspects of the patients who form the basis of this study are included in Table 1. Some of these points should be emphasized, we believe, and other observations and findings too infrequent to justify inclusion in the table are worthy of some comment.

It should be noted that although thrombosis is often regarded as a disease of the later years, 13 of the patients in this series were in the fifth decade of life when the signs of thrombosis occurred. We have been surprised to find the highest incidence in this middle-age group. It becomes evident that 40, rather than 50, is the age at which we should begin to consider this diagnosis in patients presenting symptoms or signs suggestive of non-hemorrhagic stroke. In our series, there were 15 males and 9 females; there was no essential difference with respect to the side involved. The onset in this series was usually abrupt.

As in cases reported by others, the most frequent initial complaint was of hemiparesis or paralysis on awakening in the morning. However, convul-

* Parts of this paper were presented at the meeting of the American Academy of Neurological Surgery in Portland, Oregon, in October, 1949, and at the meeting of the Harvey Cushing Society in Victoria, B. C., in June, 1952.

TABLE 1

Case No.	Patient	Age	Sex	Side	Duration of Symptoms	Paresis or Paralysis	Sensory Loss	Pain—Cranium	Activity at Onset	Convulsion	Onset	CSF Pres.	CSF Prot.	B.P.	Hb.	Systemic Disease	Remarks
1	H.M.	47	F	L.	4 days	Transitory paresis, rt.	Transitory numbness, rt.	Transitory ipsilat. eye	Sitting with head to rt. in auto	No	Sudden	—	—	140/94	16.0	None noted	2 episodes 4 days apart, each with sharp head turning to rt. Block at Cl. Lesion demonstrated at operation
2	L.O.	44	F	R.	2 wks.	Spastic lt. hemiparesis	Lt. hemihypaesthesia	No	On awakening in A.M.	Unrelated	Sudden	155	33	150/100	13.0	Mild arteriosclerosis	10 yrs. earlier had had lt. frontal cerebral scar removed because of convulsions. No attacks related to occlusive symptoms
3	P.D.	52	M	L.	1½ yrs.	Rt. weakness and incoordination	2-pt. discrimination impaired	No	Coma—? diabetic	No	Sudden	—	—	122/74	15.1	Diabetes, mellitus, 2 yrs.	On arousing from coma, complete rt. hemiplegia and aphasia for 3 days; gradual but incomplete improvement subsequently
4	V.K.	61	M	R. (L. narrowed at C2)	5 yrs.	Lt. spastic	Homonymous hemianopia	Rt. orbit radiating to occiput	Not known	Generalized	Sudden	120	50	160/100	14.5	Peripheral vessels thickened	Numbness and tingling lt. hand followed in 2 wks. by lt. hemiplegia while working
5	E.H.	42	F	L.	5 wks.	Moderate, rt.	Hypaesthesia, rt.	Bilat. frontal occipital, 3 mos.	Awoke in A.M. with paralysis, aphasia	No	Sudden bilat. headache for 3 mos.	68	22	100/70	15.0	None noted	Bilat. ptosis; ipsilat. carotid pulse in neck reduced
6	A.J.	43	F	L.	6 wks.	Moderate, rt.	Hypaesthesia, rt.	Lt. temporal	Not known. Transient recurring episodes	No	Sudden—recurring	—	—	110/75	14.0	None noted	Transient recurring episodes of aphasia, hemiparesis (rt.) and severe lt. temporal headaches
7	C.H.	48	M	R.	4 mos.	Lt. spastic, severe	Hypaesthesia, lt.	Bilat., temporal-occipital	None specified	No	Sudden	Ventriculogram		125/75	15.0	Peripheral vessels thickened	Head injury followed 1 wk. later by progressive lt. hemiparesis. Relationship questioned
8	T.H.	68	M	R.	8 mos.	Slight, lt.	No	Rt. orbit, forehead, 2 wks.	Not stated. "Fainted"	Unilat.	Sudden. Intermittent	150	75	180/100	15.5	Arteriosclerosis, generalized	Initial syncope followed by dysphasia and pain, transitory, and later by recurring convulsions
9	R.T.	45	M	R.	2 wks.	Flaccid hemiparesis, profound, lt.	Hypaesthesia, lt.	Rt. occipital and orbital	Final stroke on awakening in A.M.	Unilat.	Gradual	165	65	164/110	18.3	Thromboangiitis obliterans	Bumped head on drawer—rt. occipital and orbital pain immediately. Lt. arm numbness without weakness 1 wk. later. Focal convulsion lt. arm after 3 days. After 1 more wk., awoke with complete lt. hemiplegia. Carotid pulse, neck, reduced bilat.

TABLE 1 (*Continued*)

Case No.	Patient	Age	Sex	Side	Duration of Symptoms	Paresis or Paralysis	Sensory Loss	Pain—Cranium	Activity at Onset	Convulsion	Onset	CSF Pres.	CSF Prot.	B.P.	Hb.	Systemic Disease	Remarks
10	M.M.	64	M	R.	1 mo.	Slight lt. hemiparesis	Slight hemihypalgesia	No	Not known	Sensory attacks only. Lt.-sided	Sudden	"Normal"	22	120/80	16.3	Mod. arteriosclerosis in peripheral vessels	Admitting diagnosis—tumor. Patient well 2½ yrs. later
11	F.D.	47	M	R.	1 yr.	Lt. spastic	Astereognosis, lt.	Rt. temporal	Postconvulsive	Lt.-sided	Sudden	"Normal"	84	130/80	19.0	Retinal vessels arteriosclerotic	Slight improvement with bilat. cervical sympathectomy
12	E.C.	57	F	R.	1½ yrs.	Lt. spastic	Slight parietal loss	Lt. eye, cheek, teeth, ear	Postoperative	No	Sudden	62	32	125/80	12.5	None noted	Awoke with hemiparesis after laparotomy for gastric resection under general anesthesia. Other details not available. Paralysis worse in ensuing few days
13	P.B.	62	M	Bilat.	6 wks.	Slight weakness, rt.	Rt. numbness at onset	Suboccipital headache	Eating	Yes	Sudden	130	67	150/90	15.7	Arteriosclerosis, generalized	Pt. eating. Sudden rt. numbness and loss of consciousness followed. Subsequent repeated generalized convulsions
14	J.S.	15	M	L.	2 yrs.	No	No	Retro-orbital	Active at time of 1st gen. faint.	Currently rt.-sided	Sudden	Normal	39	130/85	14.5	None. Father died aet. 53 hypertension	Aet. 13 pt. playing hard in heat. Sudden loss of consciousness. Convulsions started 2 yrs. later
15	M.A.	51	F	L.	5 mos.	Rt. weakness, 4 wks.	Transitory rt. numbness	Yes. 1st symptom	Not noted	No	Gradual, intermittent	120	59	240/130	14.4	Vessels firm	Intermittent pain rt. jaw, neck, ear at onset. 2 mos. later rt. subj. bruit. 1 mo. before study lt. ptosis. 1 wk. later lt. jaw, ear, neck pain. 3 wks. later rt. hemianopia, weakness, paresthesias and aphasia
16	R.S.	57	M	L.	6 mos.	Rt. slight hemiparesis	No	No	Riding to work in A.M.	No	Sudden	390 erect	25	137/88	15.5	No	Sudden hemiplegia while riding to work. Unconscious 1 wk. Progressive return rt.-sided power. Only slightly persistent with hyperreflexia at time of entry
17	L.B.	41	F	L.	18 mos.	Rt. spastic hemiplegia	2-pt. discrimination reduced	At onset. Generalized	Not known	Rt.-sided 10 mos. after paralysis	Sudden	—	—	110/72	14.9	Peripheral vessels firm	Sudden generalized headache, unconsciousness followed by hemiplegia. 10 mos. later convulsions. Angiography in 2 diagnostic centers

TABLE 1 (*Continued*)

Case No.	Patient	Age	Sex	Side	Duration of Symptoms	Paresis or Paralysis	Sensory Loss	Pain—Cranium	Activity at Onset	Convulsion	Onset	CSF Pres.	CSF Prot.	B.P.	Hb.	Systemic Disease	Remarks
18	J.P.	32	M	R.	2 mos.	Lt. spastic hemiplegia	Lt. hemihypaesthesia	Suboccipital headache sev. days before paralysis	Paralysis on awakening	No	Sudden	—	—	130/85	13.0	No	Alcoholic (26 oz. spirits average daily)
19	A.D.	45	M	L.	8 mos.	Rt. hemiparesis	2-pt. and pain sense reduced, rt.	Occasional mid. and lt. frontal	Noted on awakening in A.M.	No	Gradual, intermittent	—	—	140/90	15.7	No	Worse after unilat. percutaneous arteriogram. Improved after stellate block
20	L.R.	49	M	L.	2 mos.	Episodic rt. hemiparesis, aphasia	Parietal lobe type	Severe occipital	Not known	Unrelated	Intermittent, progressive	78	78	160/100	17.4	Peripheral vessels firm	Alcoholic. Encephalopathy 3 yrs. earlier
21	E.R.	41	F	R.	18 mos.	Slight rt. spastic hemiparesis	No	Bilat. temp. occipital	Excitement at breakfast	No	Sudden. Subsequent exacer., remission	450 erect	13	180/120	13.3	Peripheral vessels firm	Lt.-sided bruit. Lt. mid. cerebral, rt. int. carotid occluded. Rt. mid. cerebral and both ant. cerebral well filled from lt. int. carotid
22	H.G.	45	F	R.	2 wks.	Flaccid lt. hemiparesis	No	Yes. See "Remarks"	Not known	Jacksonian	Sudden	—	99	132/78	12.3	Depression and personality change	Family reported 5-yr. history recurring severe generalized headache "with complete blindness," each 1½ hrs.' duration
23	J.G.	43	M	Bilat.	6 mos.	No	Transitory vertigo	Mild at crown	Vertigo on turning head to lt.	No	Sudden. Recurring	—	—	125/80	16.0	Thromboangiitis obliterans	Marked parasellar narrowing both carotids. Died 18 mos. later despite extensive sympathectomy
24	A.E.	62	M	L.	4 wks.	Lt. N. III weaker	Inf. nasal quadrant, lt. eye only	Generalized	Apparent on awakening in A.M.	No	Sudden	—	—	160/85	15.4	Diabetic	Severe sneezing episode night before. Oculomotor palsy and headache on awakening next A.M.

sions ushered in signs of the disease on 5 occasions. In 2 instances, the first evidence occurred while the patient was eating. Weakness or paralysis of the extremities was noted by 21 patients; in 8, the disturbance was slight and transient. One of the 3 remaining patients (No. 24) had some paralysis of function of the oculomotor nerve. One patient (No. 21), who had a left middle cerebral artery thrombosis in addition to a right-sided internal carotid occlusion, had a misleading weakness on the right; the internal carotid occlusion had produced no demonstrable neurological dysfunction on the left.

Sensory loss was less striking than motor loss. While several patients complained of "numbness" at the onset of symptoms, 11, or less than half, had persisting signs of demonstrable loss at the time of hospitalization. One patient had transient hemianopia, one permanent hemianopia, and one a slight cut in the nasal quadrant of the ipsilateral eye.

Headache, or head pain, at some time during the course of the disease was commented upon by all but 4 patients. The pain was usually orbital, retro-orbital, or suboccipital in location. Since the latter complaint is a common one in the age group reviewed, it is of questionable significance in this study. In several instances, however, there was definite onset of pain associated with that of other symptoms, and it is quite probable that orbital, retro-orbital and temporal pain was related to the thrombotic process. This is consistent with the observations of others. Five of the 6 patients with high cervical occlusion who are discussed below complained of transitory or persistent orbital or temporal pain associated with the onset of other occlusive symptoms.

In the general examination, the majority of patients had normal blood pressure. The blood and/or spinal fluid Wassermann was obtained in all but 1 patient and was negative in each case, though 1 of these patients gave a history of having had lues.

There were 2 diabetics and 2 patients with thromboangiitis obliterans. Eleven patients had some clinical evidence of peripheral vascular thickening. Two patients were alcoholic. Two patients reported injuries shortly before the onset of symptoms. The relationship between the injury and the thrombosis, if any, is difficult to explain.

Of 19 patients who had electroencephalograms, there was positive lateralization of abnormality in 11. Cerebrospinal fluid pressure was normal in every instance in which it was recorded. Spinal fluid protein was elevated approximately 50 per cent of the time. Pneumograms were obtained on 6 occasions; in 5, the ipsilateral ventricle was slightly larger than its fellow and in the sixth there was bilateral ventricular dilatation.

Arteriography was carried out by the open method in all but 2 instances. This afforded an opportunity to visualize the wall of the vessel and to establish the position of the needle within the common carotid artery. Usually, the bifurcation of the artery could be seen at the exposure and the possibility of a congenitally absent internal carotid eliminated. Frequently the throm-

bosed vessel could be observed at or above the carotid sinus or palpated through the open wound.*

Bilateral arteriograms were obtained in all but 6 instances. In 3 cases, the vertebral artery was also injected. Many of these bilateral studies were done prior to the appearance in the literature of the comment that there was an added risk to such investigation. We have had no complications in our bilateral series. Our only technical difficulty was encountered in a unilateral injection by the percutaneous method. This patient experienced a return of his previous symptoms during the course of the study, which was carried out under local anesthesia. The exacerbation was resolved by immediate procainization of the stellate ganglion.

Diodrast, 35 per cent in 25 cc. amount, was used in 18 patients. Of the remaining 6, Thorotrast alone was used in 4, 3 of whom had unilateral studies. In the other 2 patients, Thorotrast was injected on one side and Diodrast on the other. Anteroposterior as well as lateral views were usually obtained. We have felt that 25 cc. of 35 per cent Diodrast are essential to adequate visualization of the vascular system. This seems to be particularly important in the present problem since we feel it reduces the chance of having all the material ascend in the external carotid system even though the internal is patent, a situation that has been reported by some.

Injections of the vertebral artery were carried out to try to demonstrate the means of collateral circulation. In the 3 instances in which this study was obtained, however, no direct connection with the middle cerebral artery was demonstrated. Furthermore, injection of the patent carotid system gave good filling of the middle cerebral artery on the side of the occluded internal carotid in only 5 arteriograms. On the other hand, there were a number of instances of filling of the middle cerebral by way of the ipsilateral external carotid via the ophthalmic. This suggests that such circulation as remains in the affected hemisphere is chiefly via the latter or the peripheral collaterals through the anterior cerebral or vertebral systems.[13]

ETIOLOGY

A number of theories have been advanced to explain spontaneous thrombosis of the internal carotid artery. Arteriosclerosis, particularly with plaques interrupting the continuity of the intima at or near the carotid sinus, has had most attention.[2,4,6,7] Vasospasm[3,6,7,10] and trauma[8,9] have also been mentioned. Systemic diseases, such as diabetes, thromboangiitis obliterans, polycythemia and arteritis, have been suggested.[1] Retrograde thrombosis from intracranial aneurysm that has ruptured and subsequently thrombosed has also been discussed.[12] To speak more generally, injury to the wall of the artery or slowing of the rate of blood flow through the vessel, or both, appear to be significant contributing factors.

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* Palpation of the carotid in the neck or throat during clinical examination has misled us more often than it has helped in arriving at the correct diagnosis. Because of the danger of dislodging a thrombus in the relatively early case, this test has been discontinued as a routine item in the examination.

Compressive occlusion of the internal carotid as it passes the lateral process of the atlas has not heretofore received attention as an etiologic factor insofar as we can determine. Our notice was first drawn to this possibility from the study of Case 1, which will therefore be discussed in more detail.

CASE REPORT

Case 1. H.M., a 47-year-old female, reported that as she was riding across the San Francisco Bay Bridge gazing steadily through the car window to her right, she suddenly noted a right hemiparesis, right hemianopia, and aphasia. The disability lasted for approximately 5 minutes, after which it disappeared without residuum. Four days later under similar circumstances the same symptoms developed plus left

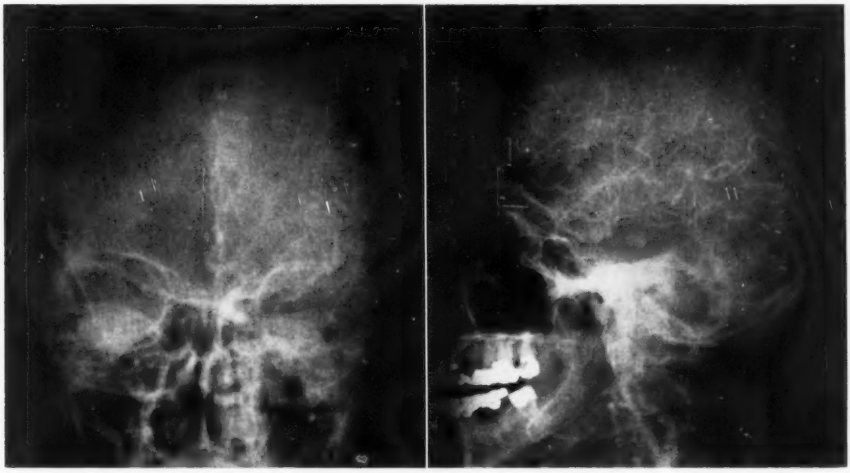

FIG. 1. *Case 1.* Anteroposterior and lateral carotid arteriograms, showing compression of the internal carotid artery at the upper cervical level.

temporal and retro-orbital pain, and a subjective left-sided bruit. She was then admitted to the University of California Hospital.

Course. Her symptoms improved. There persisted, however, a loud bruit audible over the right mastoid region, slight expressive aphasia, and minimal weakness of the right arm and face. The right deep tendon reflexes were diminished.

A left carotid arteriogram revealed absence of filling of the internal carotid artery opposite the lateral process of the atlas, both in the anterior-posterior and lateral views (Fig. 1). There was some visualization of the cavernous and petrous portions of the internal carotid, which was interpreted as resulting from collaterals through the external carotid via the ophthalmic or possibly from an incomplete occlusion of the internal carotid itself at the upper cervical level.

Operation. The left internal carotid was explored in the neck, and at the level of the transverse process of the 1st cervical vertebra, a purplish discoloration of the arterial wall was seen over a distance of approximately 3 to 4 cm. (Fig. 2). No pulsation was palpable in the discolored portion of the vessel. The artery was intimately

adherent to the lateral process of the atlas and moved with the atlas when the head was turned. The discoloration was confined to the vicinity of the point of contact of the vessel with the atlas, corresponding to the filling defect shown in the arteriogram. The adhesions binding the vessel to the atlas were interrupted and the adventitia was stripped.

Postoperative course was uneventful, and no further difficulties have occurred in the ensuing $6\frac{1}{2}$ years. The patient, however, was advised to avoid turning the head sharply to either side, and she was particularly cautioned to sleep in such a position as to prevent this occurring during the night.

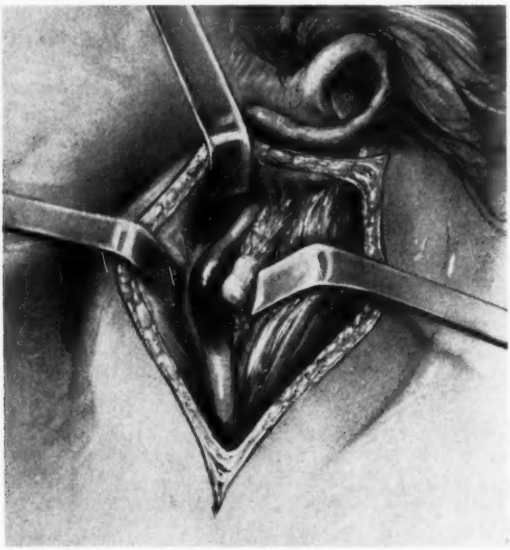

FIG. 2. *Case 1.* Drawing of the internal carotid artery as it appeared at operation. Note displacement and discoloration of the artery as it lies across the lateral process of the atlas.

Since observation of this case, we have seen 4 other patients in whom the occlusion appeared to be at the level of the transverse process of the upper cervical vertebra (Fig. 3). In addition, there was 1 case in which there was a considerable "stump" which we feel represents a retrograde thrombosis from an occlusion originally in the same general region (Fig. 4). (Wolfe[14] found the thrombosis extending to the carotid bifurcation, though earlier angiograms demonstrated the block between C1 and C2.) These 6 cases constitute one-fourth of the patients in this series.

We have also been particularly interested in noting in arteriographic illustrations of papers by others findings that are similar to our own. Thus, both Wolfe[14] and Erikson[5] presented angiograms that revealed a block in the internal carotid system at the inferior border of the atlas. In Schneider and Lemmen's[11] first case of post-traumatic internal carotid thrombosis, the same site of block is evident.

FIG. 3. *Case 7.* Representative case of occlusion of the internal carotid artery in the high cervical region.

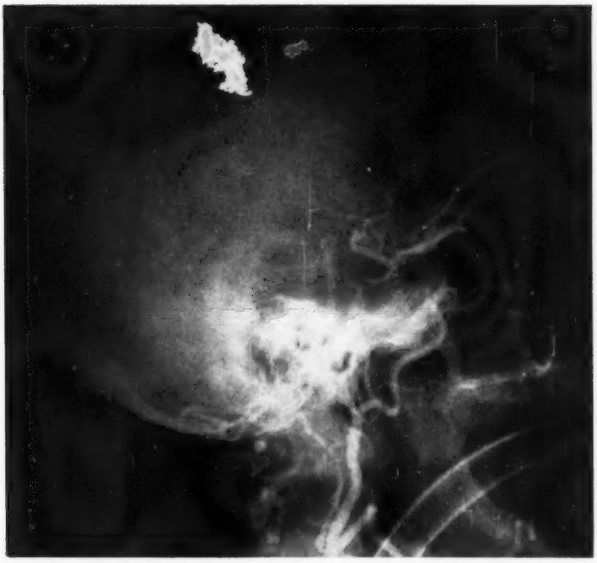

FIG. 4. Lateral arteriogram showing stump of the internal carotid, with good filling of the external carotid and the intracranial portion of the internal carotid by way of the ophthalmic and possibly the posterior communicating. Filling of the vertebral is by retrograde flow from the injection in the low common carotid.

These observations have supported a feeling we have had for some time that some extraneous factor must be participating to account for a large number of "spontaneous" thromboses of the internal carotid artery. It is recognized that arteriosclerotic plaques can of themselves underlie a thrombosis. However, when there is an exceptionally high incidence of occlusion in a particular artery or its branches, an anatomical situation which makes some area of the vessel particularly vulnerable could be responsible for some instances of the occlusive disease. Such an anatomical situation exists, we feel, in the high cervical region.

The internal carotid artery in its ascent in the neck bears a close relationship to the lateral process of the atlas. When the head is rotated to the

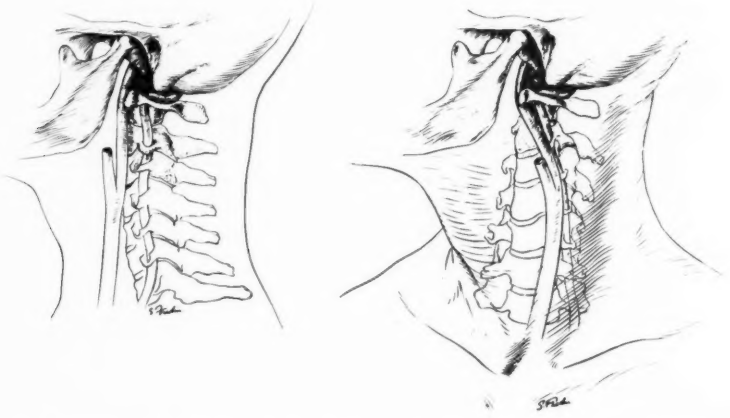

FIG. 5. Schematic representation of the displacement and compression of the internal carotid artery by the lateral process of the atlas when the head is rotated sharply. (*Left*) Normal anatomical position. (*Right*) Head turned to the opposite side.

right, the left internal carotid artery lies directly on this process; with rotation to the left, the right artery is similarly affected (Fig. 5). We have long noted the frequent presence of adhesions in the upper cervical region binding the carotid sheath to the carotid artery and to the adjacent tissues. This has been observed almost consistently in adults when there has been occasion to expose this region surgically. The adhesive process has not been seen as a rule in the younger patient. We have explained it on the basis of the close proximity of this portion of the carotid to the highly infected and frequently inflamed pharynx. We believe that it is this inflammatory thickening that accounts for the adhesion of the carotid to the lateral process of the atlas. When these adhesions interfere with the easy mobility of these two structures with respect to each other, prolonged arterial compression may injure the intima, potentially inducing thrombosis. This adhesive binding may also be responsible for the tortuous duplication so frequently observed in carotid arteriograms in this vicinity.

That occlusion can occur without a thrombosis has been observed in arteriograms when the lateral view has shown a block in the upper cervical region, yet filling has been demonstrated in the anterior-posterior view with the head turned to the normal anatomical position. Subsequent lateral views with the head turned to a lesser degree have then shown the lumen to be patent. When diagnosing a thrombosis in this region, it thus becomes evident that the possibility of temporary occlusion must be taken into consideration and eliminated by a second injection with the head slightly turned from a full lateral position or preferably by checking the filling with the anterior-posterior set of films.

Thrombosis need not occur at the actual site of compression; the retarded flow may result in clot formation at any distal point. Atlantoid compression may account for some instances of "spontaneous" thrombosis of the middle cerebral artery since this vessel is peculiarly dependent upon the ipsilateral carotid, its collateral connections being particularly devious. We believe that compression of the artery during sleep, especially with the individual prone and the head turned sharply to one side, is a real hazard and may contribute to the development of so-called hypotensive stroke where there is transitory inadequacy of circulation to the cerebrum without actual permanent occlusion demonstrable in subsequent angiograms. Compression of the carotid at this site may account for some, although obviously not all, instances of apparent carotid sinus sensitivity. It must be considered particularly when weakness or faintness accompanies strong turning of the head to either side.

It is felt that the lateral process of the atlas may irritate the carotid producing vasospasm as well as direct mechanical compression. (Embarrassment of the vertebral circulation by irritation of the vertebral arteries as they pass between the atlas and the occiput may likewise prove to be a factor in vascular inadequacy of the vertebral-basilar systems.)

When clinical evidence suggests *transitory* compression of the carotid, we have carried out surgical amputation of the lateral process of the atlas with improvement or relief of the underlying situation. This will be reported in more detail in a subsequent paper.

It should be emphasized that occlusion of the carotid at this point is presented not as the cause of all thromboses of the internal carotid artery but as a primary factor in some instances and as a contributing one in certain others.

TREATMENT

All of the patients in our series came to us relatively late in the disease. With the exception of the patient in Case 1, the minimum period between onset of symptoms and investigation was 2 weeks. Thrombectomy has not appeared justifiable in any of our cases, although it is conceivable that it might be indicated in acute thrombosis. We are aware of several instances in which the patient was under the care of a neurosurgeon at the critical period and carotid thrombectomy was immediately carried out.

In the case of occlusion apparently related to the lateral process of the atlas, if recognized early enough, freeing of the artery from the atlantoid process plus adventitial stripping may help prevent advance of the thrombotic process and possibly contribute to its subsidence. In cases of chronic occlusion, we have advised supportive treatment, with particular emphasis on the avoidance of a drop in blood pressure during sleep. In an effort to increase the collateral circulation to some degree, carbon dioxide and oxygen (10 per cent and 90 per cent) and niacin have been employed because of their reported vasodilating effect.

Many of our patients were studied during a time when there was considerable enthusiasm for stellate block and preganglionic interruption of the connections of this ganglion. Nine patients received stellate blocks. Of these, transitory benefit was apparent in 6 cases. Subsequent stellate decentralization was carried out in 5 instances including 1 of the patients with thromboangiitis obliterans. This patient received no benefit aside from a sense of improved well-being. Two patients felt that motor power increased somewhat after stellate decentralization, and 1 of them had some objective evidence of such improvement. The latter patient had a subsequent return of symptoms, which interestingly were relieved by procainization of the region from which the stellate had been removed. The relation of the stellate and its connections to the cerebral circulation and its diseases is recognized as a point of great controversy. Major benefit from either anesthetic block or excision of the ganglion is certainly uncommon; we cannot, however, say that it is non-existent, particularly when the block or ganglionectomy is employed during the acute phase of vascular embarrassment.

SUMMARY

Twenty-four cases of angiographically demonstrated thrombosis of the internal carotid in the neck, including one complete and one partial bilateral occlusion, are reported. The clinical data in this series are reviewed.

On the basis of findings in one-fourth of our cases, compression of the internal carotid by the lateral process of the atlas is presented as a possible etiologic factor in some instances of thrombosis of the internal carotid in the neck. Transitory occlusion is suggested as a potential factor in certain other dysfunctions of the central nervous system.

Some aspects of the treatment of thrombosis of the carotid artery are discussed.

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SUBDURAL COLLECTIONS OF FLUID IN INFANTS AND CHILDREN

II. STUDY WITH RADIOACTIVE SODIUM PHOSPHATE (P-32)*

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IN THE management of subdural collections of fluid in infants and children, multiple surgical intervention is often necessary. At least one of these operative procedures is performed to determine the presence of encapsulation of the subdural fluid, because removal of the capsule is necessary to effect a cure. Therefore, a nonsurgical method was sought to study the formation of the fluid and its encapsulation in relation to cause and duration of the process.

Previous studies by one of us (R. E. B.)[1] have shown that radioactive phosphorus (P-32) concentrates in significantly different amounts in pleural and peritoneal effusions. The rate of accumulation and final concentration of P-32 was closely related to the metabolic activity of the effusive process. On this basis it was thought that P-32 could be used to differentiate between subdural collections of fluid caused by various etiologies and possibly indicate the presence of encapsulation.

MATERIAL AND METHODS

The patients were unselected. The only factors governing inclusion in this series were the presence of an open fontanelle to allow needle aspiration of the subdural fluid and the reaccumulation of sufficient subdural fluid after diagnostic aspirations were made.

The plan of treatment used in this clinic is repeated aspiration of the fluid. If fluid persists after three or more aspirations, trephination is done to inspect for encapsulation. Capsules are removed by craniotomy. Trephination is performed only after 10 days of aspiration and craniotomy is performed at least 1 week after trephination if fluid is no longer accumulating or if the amount removed by each aspiration is decreasing. If bilateral, operative removals of the capsules are separated by at least 10 days.

The radioactive phosphorus study was done between the 2nd and 6th fontanelle tap and always before trephination.

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One to 200 microcuries of radioactive phosphorus (P-32) as sodium phosphate in a volume of 1 to 2 ml. was injected intravenously. Samples of subdural fluid and blood were obtained 3 hours later. Whole blood was mixed with ammonium and potassium oxalate and the plasma was separated. Subdural fluid was centrifuged. Duplicate 1 ml. aliquots of plasma and subdural supernate were placed in 2 ml. glass planchets. The planchets were brought to volume by the addition of water and then dried in the air for 24 to 48 hours. Each sample was counted in a shielded lead holder with a background of 30 to 45 CPM. Individual planchet counting rates ranged from 400 to 3000 CPM (values from 9 to 100 times background). The activity in each instance was measured with Geiger counters using thin end window mica tubes (Tracerlab TCC-2).

The activity of the subdural fluid was expressed as a number obtained from the following equation:

$$\text{Subdural activity} = \frac{\text{CPM/ml. subdural fluid}}{\text{CPM/ml. plasma}} \times 100.$$

RESULTS

In many of the cases studied, erythrocyte and leukocyte counts were made on the subdural fluid obtained for the measurement of the radioactive content. There was no obvious relationship between subdural P-32 activity and the several cell counts. In several cases in which the subdural fluid was very bloody, samples were also prepared without centrifugation. When compared to the standard centrifuged sample, the P-32 activity was increased a maximum of 3 per cent by the presence of the erythrocytes.

In the early studies samples were taken at frequent intervals. The maximum concentration of P-32 in the subdural fluid occurred at approximately 3 hours after the injection of the radioactive substance. This was confirmed in one patient in whom a catheter was inserted into the subdural space for continuous drainage. Samples of subdural fluid were obtained every 15 minutes. The activity in the various samples of subdural fluid, cerebrospinal fluid and plasma are plotted in Fig. 1. It may be seen from this graph that the peak of P-32 activity in the subdural fluid occurs 3 hours after the intravenous injection of the radioactive substance.

The results of 35 trials in 29 cases are shown in Table 1. Only 5 of these cases have not been checked by direct inspection through trephine openings and these 5 have been followed carefully for 8 months or more. Those results that are in boldface type were obtained from cases in which encapsulation was proven at operation; those in italics were obtained from patients found at operation to have a thin layer of reactive material on the under surface of the dura mater, but not to have organized capsules necessitating surgical removal.

The level of radioactivity in subdural fluid indicating encapsulation varied, depending on the etiology. As seen in Table 1 those subdural collections of fluid associated with malnutrition become encapsulated and have a

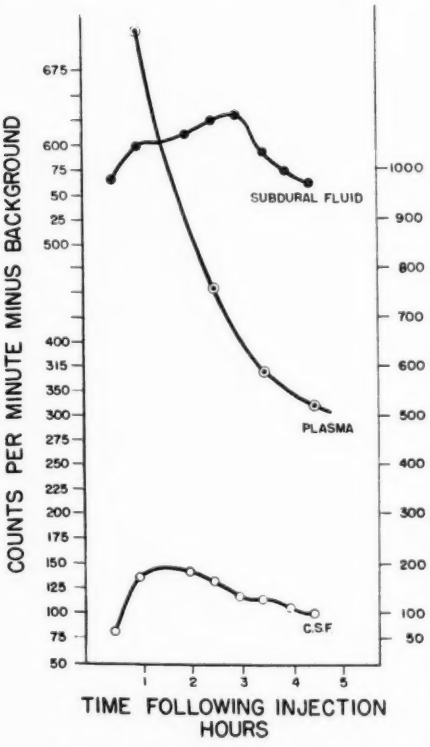

FIG. 1. Concentration of radioactive substance in the cerebrospinal fluid, subdural fluid and blood plasma.

much lower activity than those caused either by trauma or inflammatory processes. A subdural collection of fluid resulting from malnutrition or certain metabolic disorders, having a P-32 activity by this method of 15 or above, should be encapsulated. Inflammatory and traumatic subdural collections of fluid with P-32 activity levels below 120 are not encapsulated, but they are encapsulated at 130 or above. In the borderline cases, the use of trephination and inspection to determine encapsulation should be depended upon for complete safety.

In several cases it was possible to do repetitive P-32 determinations. These were cases in which there were repeated bouts of bleeding or in which the quantity of subdural fluid did not decrease despite multiple daily aspirations. (It is the rule in this clinic that prior to resection of the capsule, the amount of subdural fluid must be decreasing. This obviates the necessity of postoperative aspiration of fluid.) In one case four separate P-32 studies were done. These showed a progressive increase in the P-32 activity passing from the unencapsulated level through the doubtful and well into the encapsulated range. Surgical exploration confirmed encapsulation. In one case the first study was performed early and because of continued bleeding, aspiration of the fluid was continued for several weeks longer than in the average patient. Before operation the P-32 test was repeated. The first result was high and the second result was in the doubtful range. Although at the time of trephination a definite capsule was not seen, it was necessary 3 weeks later to remove thick capsules bilaterally because of the persistence of fluid in the subdural space. The reason for the decrease in the P-32 activity is unknown.

DISCUSSION

By this method the presence of a capsule around the subdural fluid has been accurately predicted in 10 out of the last 12 cases. In 2 of these cases, despite the results of the P-32 study indicating encapsulation, only a thin

TABLE 1

Subdural P-32 activity classified according to etiology with differentiation of encapsulated and unencapsulated collections

Nutritional	Traumatic	Inflammatory	Unknown
6 6			
7.5			
9.5			
11 **11**			
15			
20.4			
70	68		**79**
	108	113	
	118		
	126 127		
	129 129 129		
	136 *136*	**133**	
	141*		**139**
	145		
	149		
	151 151	**150**	
	156	**158**	
		160 160	
			169
	175		
	179		
	180		
		183	
	188	**188**	
	189 189	**189**	
		203	
		218	

* Not checked by trephination.
Boldface type: encapsulation proven at operation.
Italics: only a thin layer of reactive material on under surface of dura mater found at operation.

layer of reactive material was found beneath the dura mater when inspected through a trephine opening. It is probable that the openings were on the edge of the capsule. The persistence of fluid with repeated aspiration necessitated further exploration and thick capsules were found and removed. It is possible by this method, then, to save the patient a general anesthetic and about 10 days of hospital time, except in those few cases in which the P-32 activity falls in the borderline range. In these doubtful cases it is not always possible to use the P-32 activity to predict the presence of encapsulation and clinical judgment must be relied upon. Presence of a capsule determined by direct observation at the time of operation makes it possible in doubtful cases to list the various causes and partially clarify a large group of cases in which no antecedent history is obtained. It is interesting to note that 3 cases with histories of pneumonia or tuberculosis were classified as inflammatory as a result of the P-32 activity.

From the information obtained in this study it would seem that all subdural collections of fluid are effusions, resulting from a change in vascular

permeability (nutritional, inflammatory) or in response to an irritating foreign body (blood). Confirmation of this thesis is now being attempted and will be reported at a later date.

SUMMARY

1. A method of studying subdural collections of fluid in infants, using radioactive sodium phosphate, is outlined.

2. The results of this study in 35 trials are given.

3. It has been possible by this method to predict the presence or absence of encapsulation of the subdural collection of fluid if the cause is known.

4. Information concerning the etiology may be obtained by this method if the presence or absence of a capsule is known.

5. It appears probable that this collection of fluid in the subdural space is a true effusion.

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THE ROLE OF THE RETICULAR FORMATION IN THE COMA OF HEAD INJURY*

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HEAD injury has been a major problem of man for centuries. The first written account of the treatment of such appeared in the Edwin Smith Papyrus around the 17th century, B. C. Thirty-four centuries later in 1833, Jean Louis Petit was the first to recognize cerebral concussion as a separate entity and distinct from brain compression by hemorrhage. Cerebral concussion was first defined by Benjamin Bell in 1873 when he said, "Every affection of the head attended with stupefaction, when it appears as the immediate consequence of external violence, and when no mark of injury is discovered, is in general supposed to proceed from commotion or concussion of the brain, by which is meant such a derangement of this organ as obstructs its natural and useful functions, without producing such obvious effects on it as to render it capable of having its real nature ascertained by dissection." Little actual refinement in this definition of cerebral concussion has occurred in the subsequent 80 years.[13] The physiologic basis for the clinical phenomenon of cerebral concussion is not completely understood even though closed head injury is an increasingly common occurrence.

The mechanics of brain trauma were thoroughly studied by Denny-Brown and Russell[3,4,5] who established the laboratory criteria for concussion and made a distinction between acceleration and compression concussion. Holbourn,[12] studying the physical forces involved in head injury, showed that the rotational rather than linear acceleration forces are the main cause of brain damage in head injury, these forces being responsible for "contrecoup" damage, hemorrhage, and "probably" concussion. Gurdjian *et al.*[11] analyzed physical factors in concussion and demonstrated that acceleration, deceleration and compression all result in measurable elevation of the intracranial pressure. They concluded that sudden increase in intracranial pressure caused concussion and that the shorter the time duration of the increased intracranial pressure, the higher the pressure necessary to cause a concussive effect.

Investigations of the physiological basis of head injury have proceeded slowly along two lines—the measurement of biochemical changes and meas-

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† Markle Scholar in Medical Science.

urement of cerebral electrical activity alterations. Bornstein[2] as well as Tower and McEachern[16] have shown that acetylcholine appears in the cerebrospinal fluid following concussion and that it depresses integrated neuronal activity. Its effects can be counteracted by the use of anticholinergic drugs. Ward[18] showed that atropine is clinically helpful in severe closed head injuries, and Ruge[14] substantiated experimentally that atropine prevents the muscarinic effects of the acetylcholine that appears following trauma of the central nervous system.

The changes in electrical activity of the brain following head injury were studied by Walker, Kollros, and Case[17] who felt that the unconsciousness of concussion was the result of sudden intense neuronal discharge of the brain resembling a seizure discharge. Foltz, Jenkner, and Ward[6] were unable to support this conclusion since their electroencephalographic studies during experimental concussion showed flattening of the cortical electrogram and changes that were otherwise surprisingly minimal. Their subcortical recordings, likewise, showed the same depression and flattening. The one dramatic exception to this was the activity recorded from the medial reticular formation which always showed far greater and longer electrical depression than other structures studied.

In view of this finding, certain major points concerning the function of this central brain stem area are pertinent: (i) electrical potentials evoked by peripheral stimulation may be corticipetally conducted not only through the classical lemniscal system and thalamic relays, but also are conducted through this central brain stem and medial thalamus;[9] (ii) electrical stimulation in this area, from which potentials evoked by peripheral stimulation can be recorded, will cause the so-called "arousal" reaction in the dormant animal and is associated with changes in the electroencephalogram from a sleeping to a waking state;[15] (iii) electrolytic lesions of this area produced in the experimental animal cause prolonged coma and unreactivity to environmental stimuli.[8]

This central region in the cephalic tegmentum is thus intimately concerned with consciousness. Since this region receives impulses from the classical sensory pathways, it is presumed that this continuous sensory bombardment "drives" the reticular activating system and thus maintains the conscious state. Reduction in this sensory input results in spontaneous sleep from which the subject may be aroused by appropriate increase in the sensory input. Abolition of this sensory driving results in coma.

It was postulated that the unconsciousness produced in cerebral concussion could be based on changes that prevent the normal sensory "driving" of the reticular formation. This could occur by direct neuronal depression of the cells in the reticular core, or by loss of collateral afferent conduction into the central core from the main ascending sensory pathways. Since initial experimental work has shown that the total electrical activity in this area is reduced,[6] the present effort was intended to determine the effect of concussion on the reactivity of the reticular core to certain of the sensory stimuli which ordinarily "drive" the reticular formation.

METHODS

Experiments were carried out on 19 monkeys (*Macacus rhesus*). All weighed 3 kg. (plus or minus 0.2 kg.). Eleven of the animals were used as controls to gain experience and to study the recorded evoked potentials from the reticular formation during peripheral nerve stimulation. Eight identical experiments were then performed immediately after acceleration concussion to demonstrate changes in these potentials recorded from the reticular formation.

Operative procedures, including craniotomy, vein catheterization, peripheral nerve (sciatic or ulnar) exposure, and tracheotomy, were done under light ether anesthesia. Wounds were thoroughly infiltrated with procaine and 1 hour after complete recovery from anesthesia, respiratory paralysis was induced with dihydro-beta-erythroidine HBr and respirations were maintained with the Palmer respiration pump. Nerve stimulation through silver hook electrodes under mineral oil was accomplished through a Grass stimulator and isolation unit delivering recurrent single square wave pulses of 1 to 0.5 msec. duration at 2–6 V.

Through a small skull trephine, a three-electrode grid was inserted with a stereotactic instrument so as to core down through the reticular formation and medial leminiscus of the brain stem in a plane parallel to its long axis. The electrodes, insulated except at the tip, were stainless steel or tungsten wire, 0.2 mm. in diameter. The medial two electrodes were 1 mm. apart and traversed the reticular formation. Potentials were recorded by bipolar technique between these two electrodes. The single lateral electrode was 2 mm. farther lateral and fixed in the grid to be 5 mm. more advanced in depth than the medial two. Potentials were recorded between this electrode traversing the lemniscus and a deep insulated needle electrode placed well beneath the corresponding ear. Four planes of recording "runs" were made serially in the dorsoventral diameter to assure a complete sampling of the reticular activity in each case and records were taken at millimeter intervals of advance.

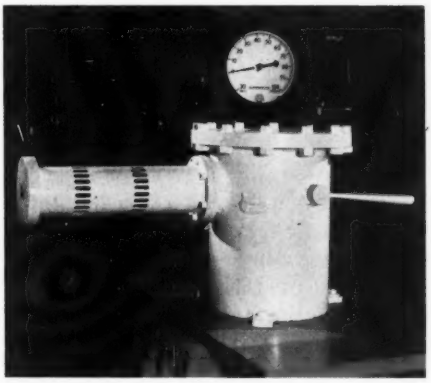

FIG. 1. Concussion gun.

Conventional biological amplifiers were used and the brain stem responses were displayed on two dual beam oscilloscopes—one for monitoring, and one for photography.

The contralateral ulnar nerve was stimulated in 2 cases and in 3 cases homolateral, contralateral, and bilateral sciatic nerve stimulation was utilized. Subsequently all animals concussed were stimulated on the contralateral sciatic nerve since this produced the most reliable evoked potentials in the reticular formation.

Production of Concussion. Since there is still controversy as to whether compression and acceleration concussion represent the same phenomenon, we preferred to use acceleration concussion. Fig. 1 demonstrates the gas pressure gun which was designed to produce acceleration concussion. It has been described previously,[6] and consists of a compressed gas chamber from which a piston is driven down the barrel

at velocities variable from 0 to 50 feet per sec. Acceleration in excess of 2500 feet per sec. is possible. An almost instantaneous blow against the animal's head is achieved by the built-in compression head on the barrel which stops the piston immediately after contact with the skull. The animal is freely suspended with skull held lightly against the baffle plate. A metal plate attached directly to the skull transmits the energy directly to the skull itself.

The end-point in each experiment for production of concussion was loss of consciousness associated with respiratory irregularity, and loss of corneal and pinnal reflexes.[3,4,5] Immediately after an adequate concussion was obtained, recording technique as in the control series was followed as rapidly as feasible. The animal was placed in the stereotactic instrument, trephination of the skull was accomplished and the electrode grid was inserted rapidly down into the midbrain and the peripheral nerve stimulation begun. The first adequate recordings were usually obtained at about 20 minutes after concussion. In 6 of the 8 animals it was necessary to use intravenous dihydro-beta-erythroidine to maintain immobility of the animal and allow adequate recording.

At the conclusion of all experiments the animals were perfused, the brains were removed intact, and serial section of the brain stem was done with Nissl and Weil stains to accurately localize the positions of the electrodes. In many cases of adequate concussion skull fracture was produced though in no instance was gross brain damage evidenced. Anatomical verification of electrode position was made in all cases.

RESULTS AND DISCUSSION

The results fall into two categories—observations on the characteristics of the sensory evoked potentials in the reticular formation and the effect of concussive head injury on these sensory evoked potentials. Fig. 2 shows the area of the reticular formation that was investigated, extending from the cephalic tegmental brain stem from the subthalamic region down to the level of the inferior olive. The blocked-in area on this diagram of the parasagittal brain stem of the monkey shows the extent of the area where evoked potentials from peripheral sensory stimulation were actually recorded. This represents a core which is 1 to $3\frac{1}{2}$ mm. lateral to the midline, and is a longitudinal reconstruction from the histological cross-sections of the brain stems.

Fig. 3 is a photomicrograph of the cross-section of the upper brain stem of the monkey showing the usual location of the grid electrodes. At this level of the superior colliculus, the three-grid electrode tracts are present in four dorsoventral planes. The medial two electrodes traversed the length of the brain stem reticular formation. The recording was bipolar between these two electrodes. The lateral electrode traversed the lateral ascending sensory pathways, the medial lemniscus, where the recording was monopolar with the reference ground lead at the base of the skull laterally. In order to obtain simultaneous evoked potentials from the reticular formation and the medial lemniscus, this electrode was 5 mm. in advance of the other two.

Our results in the control study of the sensory evoked potentials in the reticular formation support the observations of Amassian,[1] and French, Magoun *et al.*[7–10] The evoked potentials in the tegmental core contrasted

sharply with those transmitted over classical sensory pathways. Fig. 4A is an oscilloscope record of the potentials in the medial lemniscus (upper trace) and the reticular formation (lower trace) evoked by a single stimulus to the contralateral sciatic nerve. In the classical sensory pathway, the latency was short (5–8 msec.), the wave form was spike-like, and there was

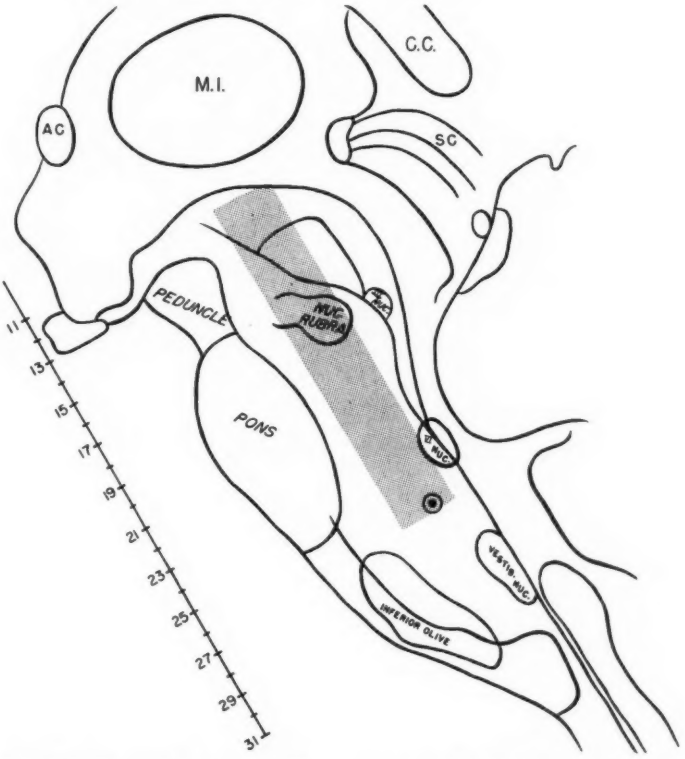

FIG. 2. Diagram of parasagittal section of brain stem of monkey. Shaded area is reticular formation where sensory evoked potentials were recorded as determined from histological reconstructions. Scale is in millimeters.

little or no attenuation on repetitive stimuli. The evoked response in the reticular core, on the other hand, showed a long latency (14–24 msec.), and a broader gross response indicating temporal dispersion. Interaction and attenuation of successive responses to multiple peripheral stimulation were likewise demonstrated. This record shows the evoked response generally found in 704 recording sites within the reticular formation in the control group.

Fig. 4B shows the typical response recorded from the same area in another animal 25 minutes after concussion. Except for the concussion, the

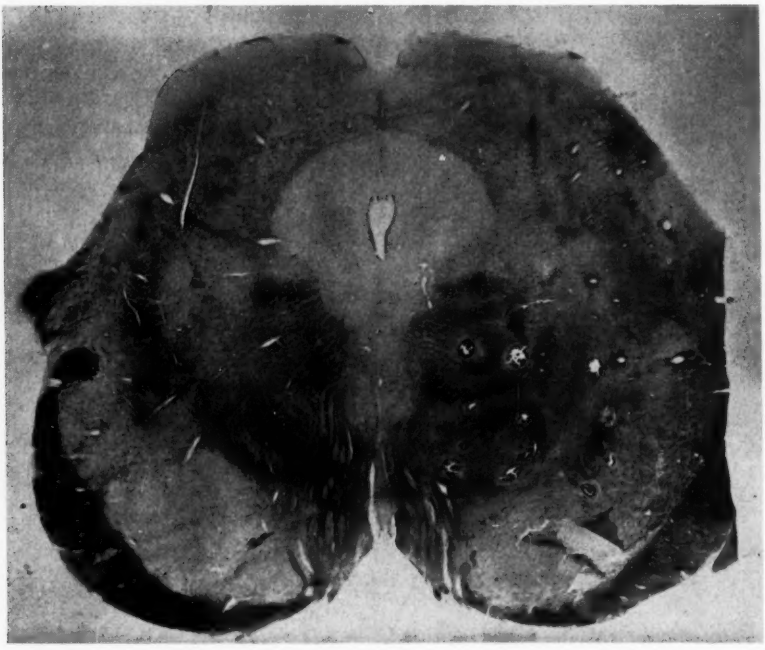

FIG. 3. Cross-section of midbrain showing recording sites of electrode grid. Weil stain.

conditions were identical. The upper trace shows the medial lemniscus evoked potential but no evoked response is present in the reticular formation (lower trace). This lack of evoked response in the reticular formation was very striking. In 6 of the 8 animals concussed, practically no sensory evoked response appeared in the reticular formation (396 recording sites). In one

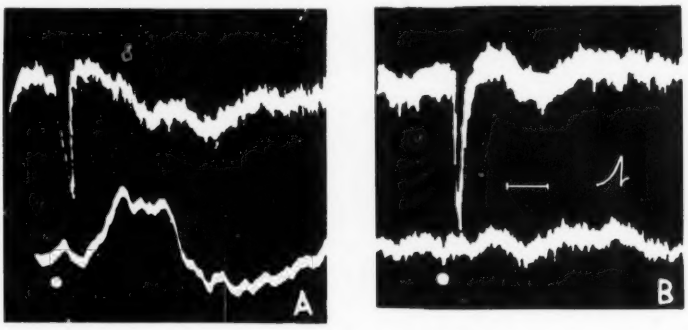

FIG. 4. Oscilloscope record of concomitant evoked potentials in brain stem reticular formation (upper trace) and medial lemniscus (lower trace). (A) Control without concussion. (B) After concussion. Time calibration −10 msec. Voltage calibration −50 μV.

animal, only temporary loss of this response appeared, and in the other animal a delayed but progressive loss of this response was obtained. The response in the medial lemniscus, however, was not significantly changed in any instance except immediately ante mortem.

The loss of the reticular response following concussion can be temporary as demonstrated in Fig. 5. In this case, the animal was followed intermittently for 24 hours after concussion had been produced. The oscillograph tracing is the record of the evoked potentials in the reticular formation

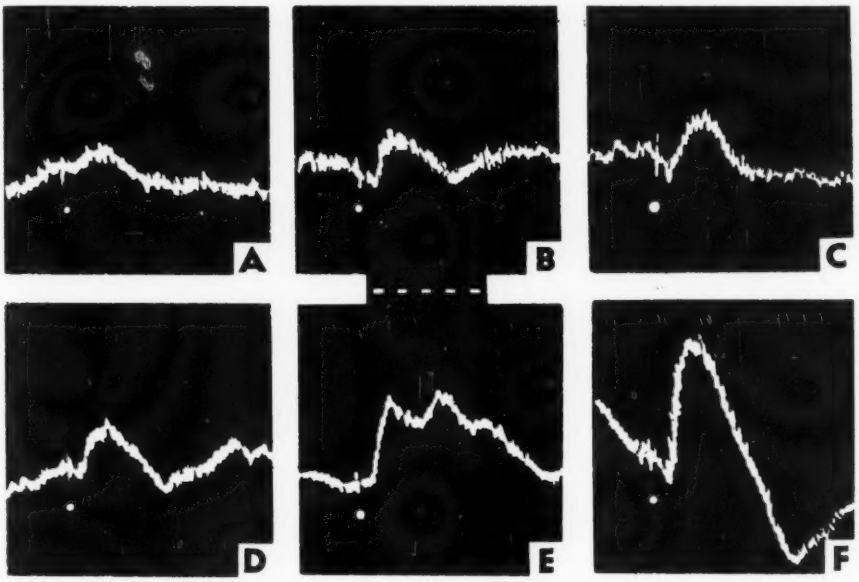

FIG. 5. Spontaneous return of sensory evoked potentials in reticular formation after concussion. Stimulus—contralateral sciatic nerve. (A) 25 minutes after concussion, (B) 1½ hours, (C) 3 hours, (D) 6 hours, (E) 9 hours, and (F) 20 hours after concussion. Time calibration—60 cycle sequence.

under conditions already described. At A, only a suggestion of a response is present 25 minutes post-concussion; at B, 1½ hours post-concussion, a small response has appeared; 3 hours (C), and 6 hours (D) and 9 hours (E) after concussion, progressive improvement has occurred. At (F), 20 hours after concussion, the response is considered normal. This return of the sensory evoked potential in the reticular formation could not be correlated with the return of consciousness under the experimental conditions necessary for these recordings, but such an assumption is at least logical.

The situation demonstrated in Fig. 5 can be correlated with the clinical case of head injury in which the mild to moderate cerebral concussion produces a variable period (1 to 8 hours) of unconsciousness or loss of environmental reactivity or adjustment. This injury is not associated with obvious neurological deficits once the sensorium clears. During recovery, the

clouded sensorium—at times associated with apparent complete unresponsiveness and near coma—can be dramatically reversed by strong, repetitive sensory stimuli. Immediately on cessation of such stimuli, however, the patient lapses back into apparent near coma. Such an "arousal" has many features similar to "arousal" achieved in the narcotized experimental animal during reticular formation stimulation.

Fig. 6 demonstrates the single case of delayed loss in reticular reactivity following concussion. This record occurred in one animal in which a relatively moderate concussion was produced associated with only a momentary apnea and flaccidity. The corneal reflex was gone for 2 minutes following

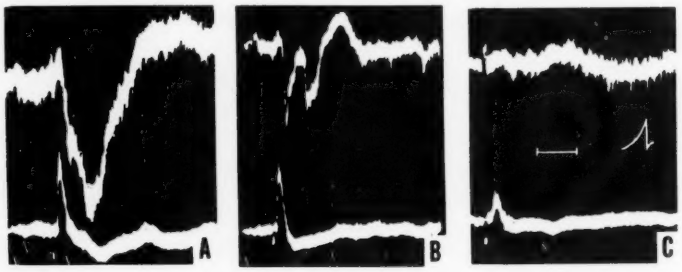

FIG. 6. Oscilloscope record of sensory evoked potential in brain stem reticular formation (upper trace) and medial lemniscus (lower trace). (A) 20 minutes, (B) 1 hour, and (C) 4 hours following concussion. Time calibration—10 msec. Voltage calibration $-50\ \mu V$.

which flailing activity required dihydro-beta-erythroidine for immobilization. At A, 20 minutes following concussion the reticular formation (upper trace) and lemniscus (lower trace) show a near normal response. At B, 1 hour following concussion, the reticular response shows diminution and change whereas the lemniscal response is essentially unchanged. At C, 4 hours after concussion, the reticular response is just barely visible and the lemniscus response is somewhat depressed. The electrodes were held in the same position during this recording. Expiration occurred shortly thereafter. There was no significant gross brain damage. It appears from this that the loss of sensory reactivity of the reticular formation in head injury may be delayed and progressive in certain cases, and unassociated with gross brain lesions.

This record may well simulate the clinical case in which the initial head injury does not appear to be severe and actual initial unconsciousness may be questioned. The patient at first appears superficially to be adaptive and reactive to his environment. Within the next 4 to 12 hours, deterioration ensues and progresses to near coma. The progressive deterioration following a so-called "lucid interval" may necessitate burr-hole exploration to rule out epidural hematoma, but none is found. The phenomenon presented in Fig. 6 adequately explains at least some of the negative explorations for hematoma. It is apparent that depression of the reticular formation can be delayed, progressive and possibly fatal.

The two types of reticular depression observed following concussion—the prompt, persisting depression, and the delayed, progressive loss—probably do not have identical mechanisms of production. The prompt loss is logically caused by direct traumatic neuronal depression in the reticular substance itself, or traumatic conduction loss in the afferent collaterals feeding in from the lateral sensory tracts. The delayed depression, however, is more suggestive of a pharmacological depression such as might be produced by acetylcholine accumulation. Such a postulate is reasonable since acetylcholine appears rapidly in spinal fluid after concussion and since these clinical cases can respond dramatically to anticholinergic therapy.

SUMMARY AND CONCLUSIONS

1. Sensory evoked potentials in the brain stem reticular formation and medial lemniscus were studied experimentally in the normal monkey and in the monkey immediately after concussion.

2. These responses in the normal animal showed constant characteristics previously described by others. The essential role of these sensory stimuli in maintaining the waking state through constant activation of the reticular formation is re-emphasized.

3. The sensory evoked responses in the reticular formation were selectively, uniformly, and dramatically absent following adequate acceleration concussion, but under proper circumstances spontaneously recovered over a period of time. The responses in the lemniscus recorded at the same time were unchanged.

4. Since unconsciousness is the single major criterion for cerebral concussion, since loss of sensory activation of the reticular formation results in unconsciousness, and since these two conditions were experimentally produced simultaneously, it is postulated that the unconsciousness produced by cerebral concussion is at least in part the result of the sudden loss of sensory activation, or "driving," of the brain stem reticular formation.

5. The delayed, progressive, fatal loss of reactivity of the reticular formation has been recorded following concussion and clinical correlations have been suggested.

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SOME FACTORS INFLUENCING THE NONVISUALIZATION OF THE INTERNAL CAROTID ARTERY BY ANGIOGRAPHY

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DURING the past two decades carotid angiography has been employed with increasing frequency to elucidate cerebral vascular phenomena. Arteriographic evidence of thrombosis of the internal carotid artery has been reported by many authors since the original paper by Moniz *et al.*[5] An extensive review of the cases reported in the literature was presented by Johnson and Walker[3] in 1951. The radiographic criterion for the diagnosis has been a failure to demonstrate filling of the internal carotid artery in the neck associated with adequate visualization of the common and external carotid arteries. Sometimes there is filling in the proximal portion of the vessel which comes to an abrupt end. This situation may be seen on successive films despite alteration in timing of exposures. However, Riishede and Ethelberg[6] have reported recently on 5 patients whose arteriograms showed no or partial filling of the internal carotid arteries bilaterally. Subsequent postmortem examinations failed to substantiate any anatomic obliteration. The common clinical feature in all of their cases was a sudden rise in supratentorial pressure with evidence of brain stem compression at the incisura. The authors postulated a diminution of cerebral blood flow resulting reflexly from embarrassment of regulatory pressor centers in the brain stem.

The purpose of this communication is to present 4 additional cases which appear to confirm the fact that certain hemodynamic factors, as yet unexplained, can produce angiographic findings similar to those seen in thrombosis of the internal carotid artery.

CASE REPORTS

Case 1. G.H., a 19-year-old youth, was admitted on Mar. 17, 1955.

Present Illness. Thirty hours previously he awoke in the morning complaining of headache; however, he went to work. One and one-half hours prior to admission he was seized with severe right head pain. Several generalized tonic and clonic convulsions followed. He was brought to the emergency room in coma.

Significant Past History. Two years before admission he was seen by his doctor for headache and stiff neck. He recovered without sequelae. No diagnostic studies were done.

Examination. Blood pressure was 140/80; pulse rate 66, and respiratory rate 28. The patient was in restless coma with a suggestive left hemiparesis and a left central facial weakness. The right pupil was larger than the left but was reactive to light.

There was a bilateral ankle clonus and a positive Babinski on the right with no plantar response on the left. Mild nuchal rigidity was present. A Grade II systolic murmur at the base was not transmitted. The fundi were normal.

Laboratory Data. Hematocrit was 41.5, wbc. 13,950 with 95 per cent polymorphonuclear cells, sedimentation rate 10, and blood serology was negative. A lumbar puncture revealed grossly bloody fluid with a pressure exceeding 600 mm. of water.

Course. The patient was observed over the next 8 hours, during which the vital signs were stable. He became responsive and said a few words. The next morning a lumbar puncture revealed a pressure of 330 mm. of water. The fluid was again

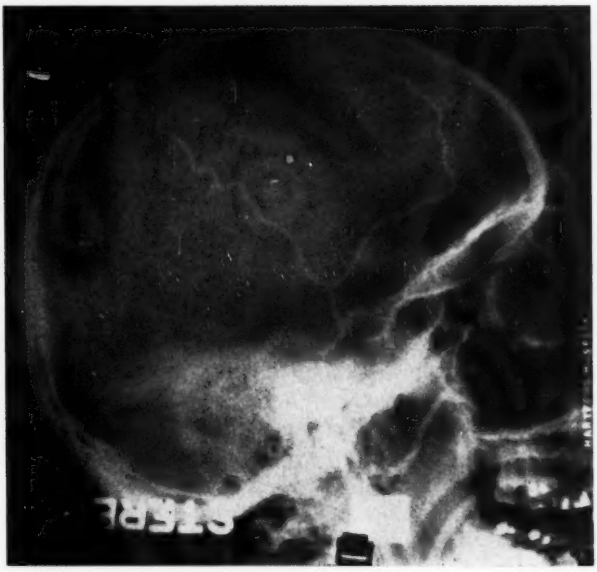

FIG. 1. *Case 1.* Right carotid angiogram.

bloody. Soon thereafter he had another generalized convulsion followed shortly by dilatation and fixation of the right pupil. The right fundus showed several retinal hemorrhages and early choking of the optic disc. He was taken to the X-ray Department where an endotracheal tube was inserted after intravenous Pentothal. A gallop rhythm developed, with a pulse rate ranging between 200 and 250. Intravenous Pronestyl caused reversion of rhythm to normal. At the beginning of percutaneous arteriography the blood pressure was 110/60 and the pulse rate ranged between 100 and 120. After a stellate block the right common carotid artery was punctured. Despite variations in the timing of exposures, consistent filling of the external carotid and common carotid was obtained with some visualization of the internal carotid in the neck but none intracranially. Consequently, the common carotid was exposed and injection under direct vision was performed with similar results (Fig. 1). Attention was turned to the left carotid vessels. At this point the left pupil became dilated and fixed. Spontaneous respirations ceased. The patient was maintained by artificial respiration via the endotracheal tube. During this time

the blood pressure and pulse remained stable. Percutaneous injection of the left common carotid artery again gave good visualization of the common and external carotid vessels without demonstrating the internal carotid artery intracranially (Fig. 2). At the conclusion of the procedure the blood pressure was 95/60 and the pulse rate ranged between 90 and 95. One hour later the patient expired. Unfortunately, permission for a postmortem examination could not be obtained.

Comment. Naturally, without benefit of an autopsy the exact nature of the pathological process remains in doubt. A ruptured aneurysm or angiomatous malformation was considered the likeliest cause of the subarachnoid hemorrhage. The failure to attain intracranial filling of the carotids after bilateral attempts points to something other than a localized obliteration of the vessels. The similarity of angiographic pattern of the two sides reinforces this impression.

FIG. 2. *Case 1.* Left carotid angiogram.

Case 2. M.M., a 36-year-old male, was admitted on July 21, 1955.

Present Illness. The evening before admission he was seized with a severe right parietal headache. He retired early and slept. The next morning he was unable to get out of bed because of left-sided weakness. There had been no unconsciousness.

Significant Past History. One year prior to admission the patient had severe hypertension following acute nephritis. Seven months before admission digitalization was necessary to combat pulmonary congestion. Later he was treated with Serpasil which brought about a decline of his blood pressure from an average of 200/118 to 160/90.

Examination. Blood pressure was 190/110, pulse rate 84, and respiratory rate 22. The patient appeared drowsy but was oriented. His speech was slurred. There was a left hemiparesis including the face, the upper extremity being more involved than the lower extremity. There was diminished sensation to pinprick over the face and upper extremity on the left. There were a positive left Babinski, increased left deep

tendon reflexes, diminished left cremasteric reflex and a left homonymous hemianopsia. The fundi showed evidence of old retinopathy; there was no choking. The pupils were equal but reacted sluggishly to light. There was a mild periorbital edema.

Laboratory Data. Hemoglobin was 12.5 gm., hematocrit 47.5, wbc. 15,500 with 91 per cent polymorphonuclear cells, sedimentation rate 32, and blood serology was negative. An electrocardiogram revealed left axis deviation, and ventricular hypertrophy with ischemic changes. Urine was normal. Roentgenograms of the skull showed no abnormalities.

Course. During the succeeding days the headache persisted and the patient be-

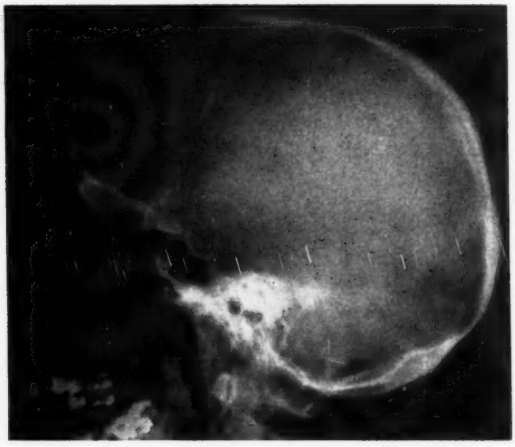

FIG. 3. *Case 2.* Right carotid angiogram (early).

came less responsive. On July 24, 1955 there was beginning drooping of the right lid. A lumbar puncture on July 25, 1955 revealed clear fluid with a pressure of 300 mm. of water. The protein was 44 mg. per cent and 1 red blood cell was seen. The patient became incontinent and eventually unresponsive. At that time his blood pressure was 230/110, his pulse rate 68 and respiratory rate 20. His right pupil became dilated and fixed, and Cheyne-Stokes respirations supervened. Using procaine, a right percutaneous common carotid puncture was made. Numerous injections of 35 per cent Diodrast at different time intervals failed to fill the intracranial portion of the internal carotid (Figs. 3 and 4).

Operation (R.H.D.). A right subtemporal craniectomy was performed. On opening the dura mater the brain was very tense and edematous but no clot was seen. An inferior right temporal lobectomy was performed followed by incision of the tentorium. This procedure was followed by a gush of normal spinal fluid from the region of the incisura. The 3rd nerve was seen to be lying freely. At the end of the procedure the brain was pulsating well.

Postoperative Course. The right pupil became reactive and less dilated. On July 27, 1955 he was speaking well. The pupils were equal and reactive. The left hemiparesis showed gradual improvement. By discharge on Aug. 9, 1955 he was able to walk with assistance.

Readmission. On Oct. 2, 1955 the patient was readmitted for postoperative angiography. His blood pressure was 160/100 and his pulse rate was 78. Examination revealed residua of his previous left hemiparesis. Injection of the right common carotid percutaneously revealed filling of the siphon with the posterior communicat-

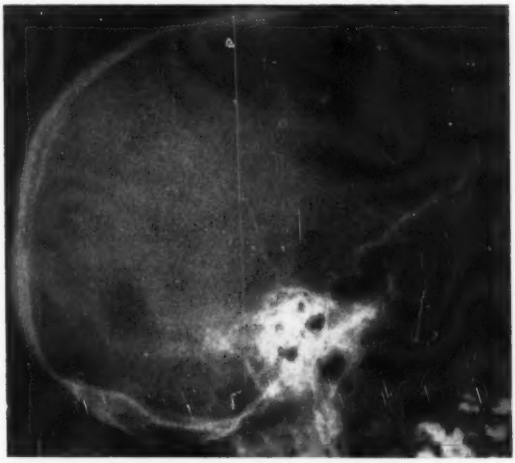

FIG. 4. *Case 2.* Right carotid angiogram (late).

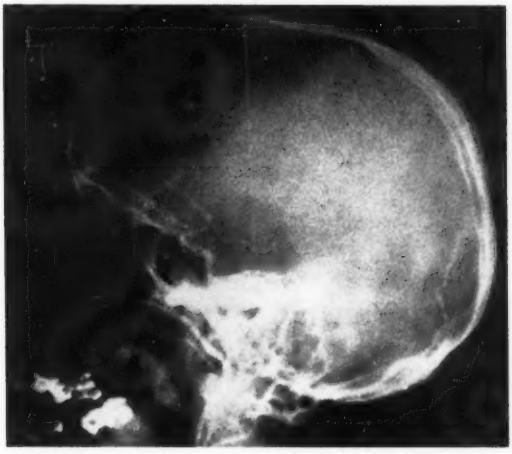

FIG. 5. *Case 2.* Right carotid angiogram (postoperative).

ing and anterior choroidal arteries being demonstrated (Fig. 5). The middle and anterior cerebral vessels were not seen. The external carotid and its branches appeared well filled.

Comment. Clinically, this patient was felt to have had a massive infarction secondary to thrombosis of the right middle cerebral artery. The post-

operative arteriogram suggests that the internal carotid artery had thrombosed near its bifurcation. The clinical and postoperative angiographic patterns bear a marked similarity to a case reported previously by King.[4] Another cause, however, must be sought for the failure to demonstrate the upper cervical and intracranial portions of the internal carotid in the original arteriogram. One might postulate that a thrombosis of the undemonstrated portion of the vessel existed with subsequent recanalization accounting for the apparent patency in the second arteriogram; however, the short interval of time between studies seems to cast doubt on this thesis.

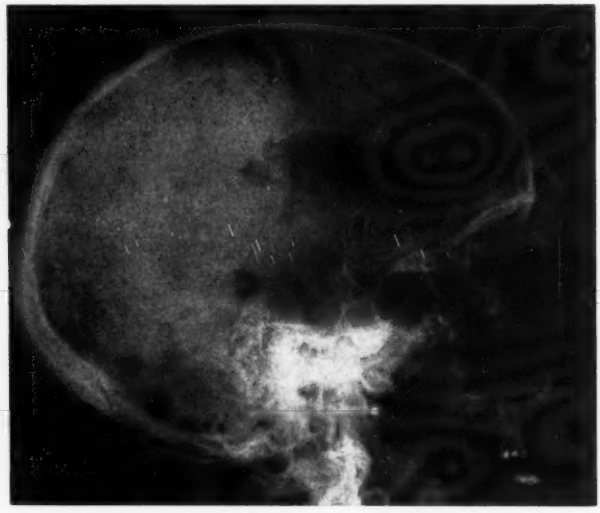

FIG. 6. *Case 3.* Left carotid angiogram.

Case 3. C.S., a 52-year-old laborer, was admitted on Sept. 1, 1955.

Present Illness. The patient entered with a 2-day story of weakness and severe headache occurring that morning.

Examination. He was found to be lethargic and unable to answer questions. His respirations became intermittently Cheyne-Stokes in type. The pupils were equal and sluggishly reactive to light. There was no nuchal rigidity. The blood pressure was 184/115 and the pulse rate was 48.

Course. Shortly after admission he became comatose with dilated fixed pupils. There was a bilateral positive Babinski sign. The fundi showed evidence of venous engorgement. The blood pressure was 180/110, pulse rate 58 and respirations were 48 and Cheyne-Stokes in type. Roentgenograms of the skull and chest were normal.

Operation (N.H.H.). Bitemporal burr holes were made revealing a very tight brain with evidence of an extensive subarachnoid hemorrhage. The ventricles were tapped, yielding fluid under extremely high pressure. Air injected revealed a dilated system with no mass lesion or shift. No catheter was left in the ventricle and the air was not released. With the blood pressure 180/90 and the pulse rate 60, bilateral percutaneous carotid arteriography was performed with 35 per cent Diodrast. De-

spite variations in the timing of exposures the common and external vessels filled with only a small portion of the cervical internal carotid being visualized (Figs. 6 and 7). At the conclusion of the procedure the blood pressure was 160/90 and the pulse rate was 90.

Postoperative Course. The patient expired 5 hours later.

Autopsy. A ruptured aneurysm was found at the origin of the posterior communicating artery on the right. The carotid arteries were of normal caliber and patent. There was a bilateral temporal pressure cone. A defect in the roof of the carotid canal was noted.

Comment. The angiograms in this case were almost identical on the two sides. Since no anatomic obliteration was found it must be concluded that

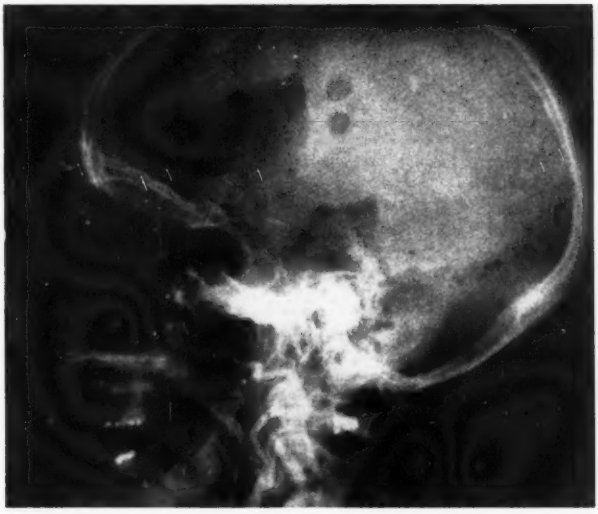

FIG. 7. *Case 3.* Right carotid angiogram.

factors affecting the cerebral blood flow were responsible. The bony defect in the middle fossa over the carotid canal brings up the possibility of direct compression of the carotids by the intracranial contents.

Case 4. D.P., a 23-year-old female, was admitted on Feb. 10, 1953.

Present Illness. The patient awoke 3 days previously with a severe frontal headache associated with nausea and vomiting. These symptoms subsided until the day of admission when they reappeared with excruciating severity, followed by the patient's collapse.

Examination. Blood pressure was 130/76, pulse rate 56, and respiratory rate 24. The patient was deeply comatose. The neck was stiff. There were occasional extensor spasms of the extremities; otherwise, she had a flaccid paralysis. The pupils were dilated and fixed to light. The fundi were normal.

Course. A twistdrill ventricular puncture was attempted immediately. No fluid was obtained but some semisolid blood clot and edematous brain extruded from

the openings. A lumbar puncture revealed a pressure of 580 mm. of water; the fluid was pink. A tracheotomy was performed followed by carotid angiography. At this time the blood pressure was 135/80, pulse rate 130 and respiratory rate 30. A percutaneous puncture on the right with injection of 35 per cent Diodrast yielded evidence of a shift of the right anterior cerebral artery to the right. An open angiogram was attempted on the left. The filling of the common and external carotid vessels was good. The proximal portion of the internal carotid was visualized but no intracranial filling occurred despite numerous attempts (Fig. 8). Six hours later she expired.

Autopsy. There was an extensive subarachnoid hemorrhage with rupture into the subdural space, predominantly on the left. There was an area of softening in the

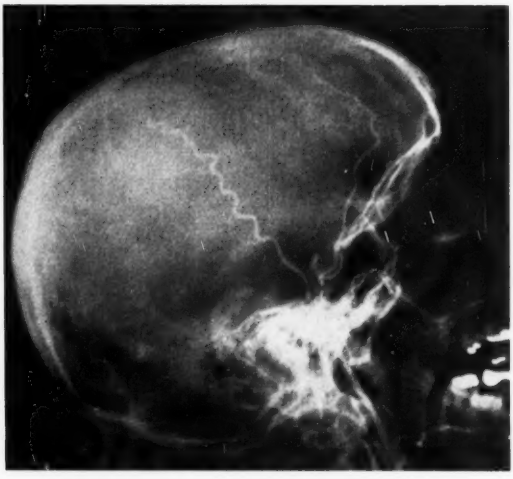

FIG. 8. *Case 4.* Left carotid angiogram.

left hippocampus overlain by a large clot, suggesting that this may have been the site of the bleeding vessel. A careful search, however, failed to divulge the presence of an aneurysm or ruptured vessel. In cut section the brain was edematous with small ventricles and there was a shift of the ventricular system to the right. There was blood in the 4th ventricle but not elsewhere in the ventricular system. Microscopic examination revealed only hemorrhagic edematous cerebral tissue. There was no evidence of thrombosis within the intracranial portion of the left carotid artery or its branches. The cervical portion was not examined.

Comment. This case is interesting in that only one side of the carotid system was visualized. The diagnosis of a space-occupying lesion on the left was made in view of the rightward shift of the right anterior cerebral artery. Despite the incomplete postmortem examination of the carotid on the left, this hardly represented a clinical picture simulating thrombosis.

DISCUSSION

The cases cited above share several features: (1) Increased intracranial pressure had occurred rather precipitously: (2) the patients were moribund,

in deep coma, at the time of angiography; (3) it was impossible to demonstrate a contrast medium, using standardized technique, within the intracranial portions of the internal carotid arteries; (4) all patients suffered from some disease of the vascular system of the brain.

Although the pathological processes responsible were not identical, the rapid rise in intracranial pressure produced definite brain stem compression in Cases 2, 3 and 4. There are strong reasons to support the assumption that similar circumstances existed in Case 1 as well. The compression of vital brain stem centers undoubtedly accounted for the state of extremis presented by all 4 patients. Failure to attain sufficient dye concentration could have been related to alterations in cerebral blood flow. Riishede and Ethelberg[6] felt that sudden severe compression of the pons, midbrain and diencephalon leads to a depressor reaction with a resultant drop in cerebral blood flow. Though certain inferences can be made from experimental studies by Ryder *et al.*[7] and Evans and co-workers,[2] no actual reflex pattern has been proven. The problem of evaluating data obtained in their 5 cases was pointed out by Riishede and Ethelberg;[6] namely, that blood pressure readings at the time of angiography were taken from the brachial rather than the carotid arteries. Furthermore, in those instances in which the intraventricular pressures were recorded, these were not taken simultaneously with the blood pressure. Several possibilities exist to account for this phenomenon. Since angiography depends upon a sufficient concentration of dye within a given vessel for radio-opacity, we cannot assume that there was no patency of the vessel; only that there was insufficient dye to be visualized by radiographic techniques. A sympathetic response causing dilatation of the external carotid system might well shunt the dye to a large degree into this system and hence fail to "fill" the internal system. Since this did not occur in 150 cases of known spontaneous subarachnoid hemorrhage,[1] in many of which there was also sharply increased intracranial pressure and some of which were terminal, it seems unlikely that this is in any way characteristic of the disease.

The possibility that these 4 cases simply represent a statistical percentage of nonfilling seems unlikely in view of their other similarities.

A relatively small proportion of the nerve supply of the intracranial circulation is estimated to be intrinsic. The remainder is in response to dynamic factors in reference to the body as a whole. It seems reasonable to assume that these reflex phenomena are mediated by way of the sympathetic and parasympathetic ganglia in the diencephalon. Therefore it seems feasible to suspect that compression of this structure may initiate a neural reflex which so alters the intracranial circulation as to make its visualization impossible.

Several extracranial causes of nonfilling ought to be considered. In some instances a very generous external circulation might tend to gain the majority of the circulating contrast medium. Conceivably, a stellate block might accentuate such a phenomenon; however, in Case 1 a block on the right did not produce a picture appreciably different from the unblocked left side. Mechanical stimulation does not seem likely.

Obviously, more controlled experiments are necessary to advance the discussion of etiology beyond the status of conjecture. From the practical point of view it would seem to be important to perform bilateral angiography whenever one side suspected of being involved fails to show adequate filling. This is not without danger, however, as pointed out by Johnson and Walker,[3] if actual carotid thrombosis is present on the initial side of angiography.

SUMMARY

Four cases of nonfilling of the intracranial portion of the internal carotid arteries angiographically are presented. The failure to demonstrate obliteration of the vessels is emphasized in order to distinguish it from the picture seen in thrombosis of the internal carotid arteries in the neck.

The reproductions of the angiograms by Dr. Francis G. Reilly, Hartford, Connecticut, are gratefully acknowledged.

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SUBARACHNOID HEMORRHAGE: PROGNOSTIC FACTORS

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THE problem of spontaneous subarachnoid hemorrhage has been receiving much attention during the past few years. This has been particularly true of aneurysms as the chief etiological agent. Prognosis in the cases of ruptured intracranial aneurysm seems to be well established. The present study is concerned chiefly with those cases in which the diagnosis of aneurysm could not be made by conventional techniques.

Statistical surveys emanating from various clinics must be evaluated with full knowledge of certain variables. A disease that may be suddenly fatal has a certain natural selectivity which prevents a large diagnostic clinic from seeing its proportionate share of the acute problem. The small hospital with limited facilities cannot in many instances arrive at a definitive diagnosis. The philosophy of the community physician is also a major factor in the diagnosis of this disease and finally, the attitude of the neurosurgeon and his past experience will influence his handling of a given case. Our statistics can reflect, therefore, only those of our medical community.

During the past 10 years 151 patients with spontaneous subarachnoid hemorrhage have been admitted to the Hartford Hospital. There were 70 cases of verified aneurysms and 81 cases in which no aneurysm was demonstrated. These figures seem somewhat disproportionate to other series[1,2,9] and may be ascribable to technical difficulties with angiographic techniques in the early cases as well as to a number of aged patients in whom angiography was deemed ill advised. Recent figures would indicate a somewhat higher percentage of subarachnoid hemorrhages with demonstrable aneurysms.

Little has been reported about the fate of patients with subarachnoid hemorrhage in whom no definite evidence of an aneurysm was found. With this in mind a follow-up study was made of the 81 such patients in our series to learn whether they had survived an appreciable time and if not, whether they had died as a result of subarachnoid hemorrhage.

It was possible to obtain a satisfactory follow-up report, either by direct contact with the patient or more rarely by inquiry of the family physician, in all but 10 of the 81 cases (Table 1). The latest reports have all been obtained within the past 6 months. There were 52 patients still alive from 1 to 10 years following the hemorrhage, and in general they were doing well. There were 19 deaths. The over-all mortality in this group of spontaneous subarachnoid hemorrhage is about 27 per cent which compares very favor-

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TABLE 1

Summary of end results

Result	Period of Survival		
	1–5 Yrs.	Over 5 Yrs.	Total
Alive	34	18	52
	Under 1 yr.	Over 1 Yr.	Total
Dead	18	1	19
Grand Total			71

ably with the known mortality of 50 per cent in cases of aneurysm in the first year after hemorrhage.[3-5, 8, 10]

Admittedly, the follow-up period in some of these cases is too short to give accurate statistics in terms of life expectancy; nevertheless, a significant number of patients have survived a surprisingly long time. Eighteen have been followed for over 5 years and have had no further difficulty.

The presence or absence of aneurysm had to be established by existing clinical standards. We were primarily interested in those cases in which an aneurysm was not demonstrated so that no practical therapy could be instituted other than the usual routine of bed rest and repeated lumbar punctures. We did not include those cases in which the patients were admitted in extremis and an aneurysm was found at autopsy, even though in certain of these angiography had not revealed the source of bleeding. All but 6 patients included in this series had angiograms; most of them had bilateral carotid angiograms, a few had vertebral as well, and in some of the earlier cases only a unilateral angiogram was done on the side indicated by focal neurological signs (Table 2). It seems unlikely that repeated angiograms will subsequently reveal the abnormality as evidenced by the few cases in which these were done. On the basis of these studies it would seem that in the face of a spontaneous subarachnoid hemorrhage, angiography should be attempted. If it be negative, the prognosis in a given case is significantly better than in the case in which an aneurysm is demonstrated. The latter, untreated, results in a forbidding mortality.

The mortality statistics are also worthy of consideration. All but 1 of the deaths occurred within 1 year. All were caused by repeated hemorrhage except 1, and in this case the patient had a known hypernephroma of the kidney with metastasis and presumably his subarachnoid hemorrhage was

TABLE 2

Angiography

Bilateral carotid and vertebral	4
Bilateral carotid	30
Unilateral carotid	12
None	6

secondary to a metastatic lesion though he died of pulmonary metastasis. While but few postmortem examinations were performed in this group, there were 3 cases in which no aneurysm was demonstrated at autopsy though extensive subarachnoid and intracerebral hemorrhages were present (Table 3). This is not too surprising in view of the experience of others.[7] There seems little doubt that in most of these cases there were aneurysms that had failed to fill in routine angiography, and the short life-expectancy seems to confirm this impression.

The problem of aneurysms per se has been deliberately withheld as has the examination of angiomas and they will be the subject of a later communication. Aneurysms are not necessarily fatal though they carry a high mortality. More refined medical and surgical technique will decrease this mortality in time. This is particularly true in the case of aneurysms not

TABLE 3

Causes of death

Verified by autopsy	
Aneurysm	1
No aneurysm	3
No autopsy	
Recurrent hemorrhage	14
Other causes	1
Total deaths	19

diagnosed by present techniques. The 1 case in which an aneurysm was demonstrated post mortem proved to be an internal carotid aneurysm in a young male which could have been treated with reasonable chance of success had the lesion been diagnosed upon admission.

The role of hypertension has been difficult to evaluate in this series because many patients were admitted in a state of hypertension caused by the cerebral insult rather than antecedent factors. However, when a rigid criterion was established in which hypertension had been determined as a clinical entity prior to hemorrhage there were 7 patients who survived the hemorrhage: 2 are still alive and doing well 5 years later, 1 is still doing well 3 years later, and 4 died. The statistical difference here does not seem significant except that there were relatively few patients with hypertension in the series. This may well be a false evaluation since many such patients may have died at home or a diagnosis of thrombosis rather than subarachnoid hemorrhage may have been made.

The morbidity in terms of neurological deficit is another facet of this problem that has seldom been dealt with in the literature. It is of little benefit to the patient to have him survive only to live a vegetative existence because of profound neurological deficit. In the group of survivals in this series, 45 patients have resumed normal life and show no residuals at all.

Four patients without neurological deficit are under medical care for hypertension but all carry on normal activity within the limits of their hypertension. Two patients have sufficient deficit to make normal life impossible but both are cared for at home without special facilities. One patient had had a psychotic episode requiring institutionalization. This antedated the hemorrhage and reappeared 2 years following the bleeding episode. The 10 cases in which no follow-up report could be obtained represent a problem. Two of these patients had such neurological deficit at discharge that they were transferred to nursing homes where contact with them was lost. The remainder

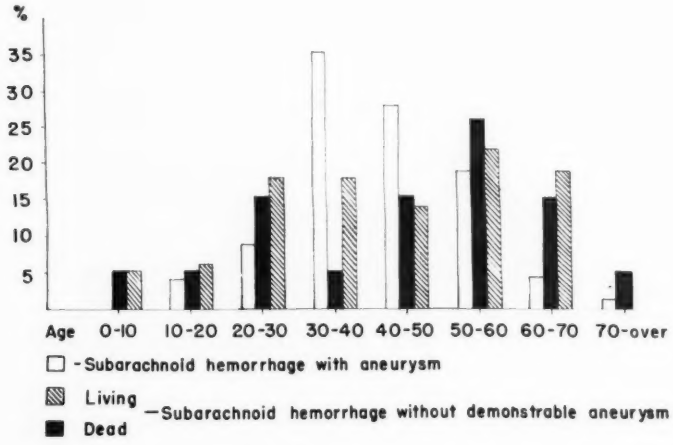

FIG. 1. Age incidence.

were either from distant cities or of such economic status that they were impossible to locate by usual methods.

The previous statistics would seem to imply that upon occurrence of a spontaneous subarachnoid hemorrhage, a careful search should be made to determine the presence or absence of demonstrable aneurysm as the etiological agent. Should it be impossible to demonstrate a causal lesion, the prognosis from the standpoint of hemorrhage is fairly good and if the person survives 1 year the prognosis is excellent. This may be one factor that explains divergent statistics reported previously.[1,6] It thus becomes obvious, in regard to prognosis, that series treating subarachnoid hemorrhage as a gross entity are by no means comparable to series of proven berry aneurysms.

In the present study there was no predilection for any age group. Most of the patients were adults (20–60 years) and there was no tendency for cases to predominate in a specific decade. Nor was there any major difference in age groups of those who survived and those who died. Comparing this group with a series of 70 verified aneurysms, the only difference seems to be that in cases of aneurysm the greatest number fall into the 30–40 age group (Fig. 1). It is interesting that all of the children included in this series have

done extremely well. One child who had a serious neurologic disability secondary to the hemorrhage has had a complete remission and is now participating in all activities in college and is actually doing ballet.

Three patients among the 52 survivors had recurrent hemorrhage; however, they recovered and have lived up to 5 years following the second attack. Presumably these, too, have aneurysms that are either microscopic or unlocalizable by angiographic techniques. The etiological factors in these subarachnoid hemorrhages are not clear but we have assumed that in most instances they represent aneurysms that have developed sufficient clot to prevent their filling with contrast medium at angiography and usually go on to complete fibrotic occlusion. A few must either hemolyze or simply fail to fill with the contrast medium during the original study either because of poor timing or the position of the aneurysm. We must also be aware of those lesions that fill only by vertebral angiography. Technical difficulties and fear of complications as well as a feeling that most aneurysms of the posterior fossa do not lend themselves to surgical treatment have tended to reduce the number of vertebral angiograms done.

SUMMARY

1. Eighty-one cases of spontaneous subarachnoid hemorrhage were reviewed in which no diagnosis of ruptured aneurysm could be made.

2. The over-all mortality was about 27 per cent.

3. The statistics would indicate that in cases in which no aneurysm could be demonstrated, the prognosis was good. If survival exceeded 1 year, it was excellent, with almost 100 per cent recovery.

4. There are some aneurysms that are not identifiable by standard angiography.

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OLIGODENDROGLIOMAS IN THE YOUNG-AGE GROUP

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OLIGODENDROGLIOMAS are comparatively rare brain tumors in the first and second decades of life. Most often they are slow-growing tumors involving the cerebral hemispheres of adults.[1]

During a recent survey of brain tumors two unusual varieties of this neoplasm were encountered. One arose near the third ventricle of a 5-year-old boy; the other involved the left frontal lobe of a 14-year-old girl. In both instances there was an initial history of trauma which obscured the correct diagnosis. The clinical and pathologic aspects of the following cases present several interesting features.

REPORT OF CASES

Case 1. B.W., a 5-year-old boy, was admitted to the hospital because of vomiting and headache. One month prior to admission he had fallen from a height of about 12 feet. He did not lose consciousness, and the only apparent residual effect was a slight limp of the left leg.

On the morning of admission he had an episode of non-projectile vomiting, which was followed by severe frontal headache. Within a few minutes he complained of abdominal pain and became drowsy. On the way to the hospital he had generalized convulsions, which lasted 5 minutes. He then became comatose.

Past history disclosed normal growth and development up to the onset of the present illness.

Examination. Temperature was 98.3°F., pulse rate 76, respiratory rate 24, and blood pressure 116/68. The patient was a well developed and nourished boy, who was unconscious. There were slight generalized twitchings of all extremities. The pupils were fixed, widely dilated, and did not react to light or in accommodation. Occasional strabismus of the left eye was noted. Funduscopic examination disclosed bilateral papilledema. Respirations were irregular, but there were no heart murmurs. There was almost constant spasm of the extremities. The slightest stimulus caused marked extensor spasm. Deep tendon reflexes were exaggerated. Bilateral positive Babinski, Gordon, Oppenheim, Chaddock, and Hoffmann signs were noted. The superficial abdominal and the cremasteric reflexes were absent.

Laboratory Data. Routine urinanalysis and blood counts were within normal limits. Roentgenograms of the heart and lungs were unrevealing. Roentgenograms of the skull disclosed no intracranial calcification, and the sella turcica was not enlarged.

Hospital Course. The day after admission there developed respiratory distress

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with cyanosis, and repeated episodes of vomiting. In spite of oxygen therapy and repeated aspiration the patient expired on the 2nd hospital day.

Autopsy. There was diffuse atelectasis of the right lung, and focal atelectasis of the left lung. The significant findings were confined to the central nervous system. The weight of the brain was 1350 gm. There was moderate cerebral edema, with a pressure cone over the brain stem, and herniation of the cerebellar tonsils. A yellow gelatinous mass, 2.5 cm. in diameter, was observed just anterior to the optic chiasm, and arising from the floor of the 3rd ventricle. On coronal section, this mass appeared to originate from the posterior portion of the 3rd ventricle, and also involved the

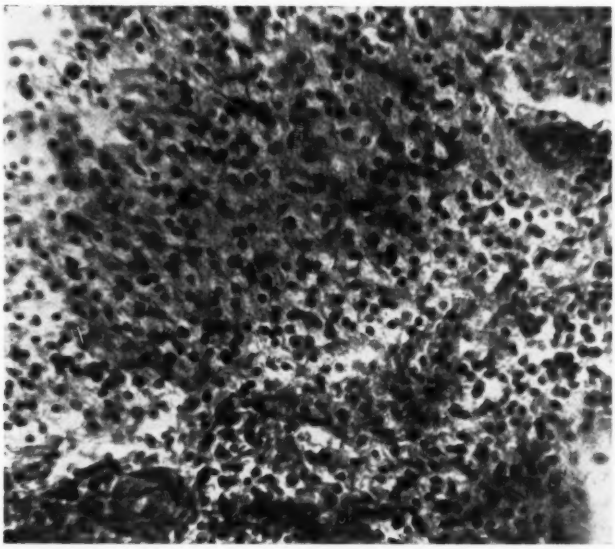

FIG. 1. *Case 1.* Oligodendroglioma of third ventricle showing small uniform cells with clear cytoplasm. Occasional thick-walled blood vessels are prominent. Hematoxylin and eosin, ×240.

lateral portions of the basal ganglia near the globus pallidus. Foci of hemorrhage and necrosis were noted throughout the cut surface of the tumor. The 3rd and 4th ventricles were markedly dilated and filled with recent hemorrhage.

Microscopic Examination. The tumor was composed of a uniform cell type. The individual cells had round central nuclei, and a clear cytoplasm (Fig. 1). Mitotic figures were rare. Slight focal calcification was present about the periphery of the tumor, and thick-walled blood vessels were prominent in some regions. The initial impression was that this represented an oligodendroglioma arising from the 3rd ventricle. Since this appeared to be an unusual neoplasm in a child of this age, representative sections of the tumor were submitted to the Armed Forces Institute of Pathology. The members of this institution confirmed the original impression of oligodendroglioma.[2]

Case 2. P.S., a 14-year-old white girl, was admitted to the hospital because of convulsions. Two and one-half months previously she had fallen at a roller skating rink. She was unconscious for a few minutes, but did not feel ill. Upon returning

home she had a convulsion, and was then promptly admitted to another hospital. The convulsions were temporarily controlled with Dilantin, and she was discharged. Upon her return home, she complained of headache, and had repeated episodes of vomiting and convulsions. She was readmitted to the same hospital the next day, at which time complete blindness and ptosis of the left eye were noted. Examination disclosed no movement of the left eye, and a complete 2nd and 3rd nerve palsy on the left. Lumbar puncture revealed normal pressures. Chemistry studies and the cell count of the spinal fluid were within normal limits. She continued to complain

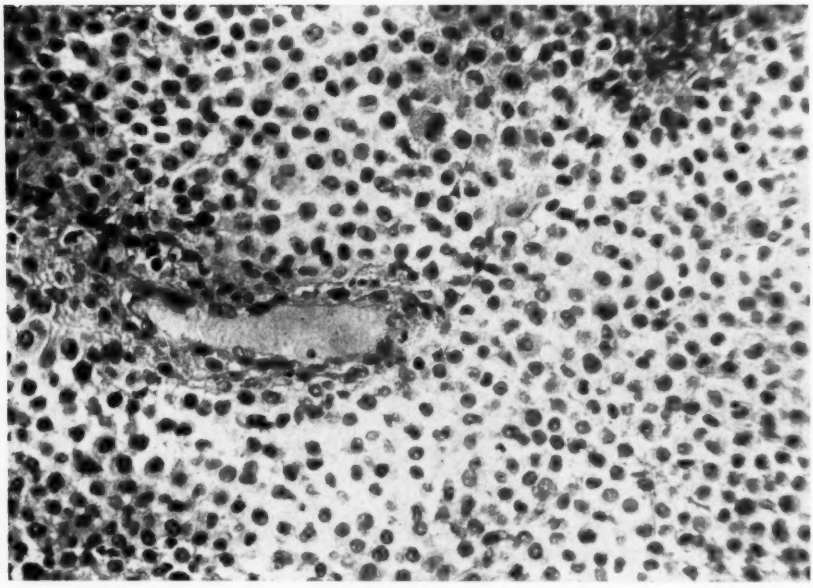

FIG. 2. *Case 2.* Oligodendroglioma of left frontal lobe showing the same type of cells as in Case 1. Hematoxylin and eosin, ×340.

of headache, and at times was stuporous. She was then transferred to the Robert Packer Hospital.

Examination. Temperature was 98.6°F., pulse rate 85, respiratory rate 24, and blood pressure 110/70. The patient was a well developed and nourished young girl, who appeared acutely ill. She complained of severe generalized headache. The head was tender to percussion, the neck was stiff, and a positive Kernig's sign was present. She had a complete left 3rd nerve palsy, right facial weakness, and definite weakness of the right arm and leg. Deep tendon reflexes on the right were hyperactive.

Laboratory Data. The rbc. count was 4,200,000, with 12.0 gm. of hemoglobin per 100 cc. The wbc. count was 28,000, with a differential of: polymorphonuclear leukocytes 93, and lymphocytes 7. Urinanalysis was negative.

Hospital Course. Shortly after admission a carotid arteriogram was done. Diodrast was injected into the left internal carotid artery, and there was good visualiza-

tion of the major branches. Slight medial displacement of the ascending branches in the mid-line was noted.

1st Operation. Multiple trephines of both frontal and temporal regions, and of the left parietal region were performed. The brain was tense, but no mass was detected. However, a catheter was placed in the right lateral ventricle. During the procedure the patient's condition was critical, and her respirations were extremely labored.

Course. Within 12 hours after operation her respirations ceased. Artificial respiration was started, and after intubation, spontaneous but irregular respirations were observed.

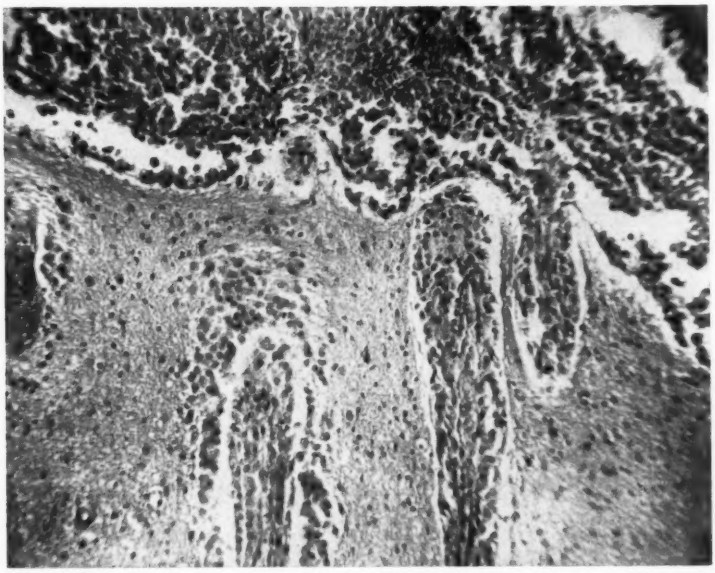

FIG. 3. *Case 2.* Broad sheets of tumor cells are present within the subarachnoid and perivascular spaces. Hematoxylin and eosin, ×160.

2nd Operation. A large frontal flap was turned down. The brain was tense and herniated through the operative defect, requiring removal of almost the entire left frontal lobe. At the base of the frontal lobe was a large firm mass, which also involved the adjacent temporal and parietal lobes. Along the sphenoid wing there appeared to be local infiltration of the dura mater. Although the tumor was considered hopelessly inoperable, as much as possible was removed in an attempt to provide decompression.

Course. Following surgery the patient was unresponsive, and she died within 6 hours.

Pathological Report. The operative specimen consisted of three portions of frontal lobe, the largest measuring 8.0 cm. in greatest diameter. The inferior surface of each portion was infiltrated by a hemorrhagic, friable, gray-white neoplasm. Histologically this tumor (Fig. 2) and that observed in Case 1 had almost identical features.

Autopsy. The pertinent findings were confined to the brain, the weight of which was 1350 gm. The gyri were flattened and the sulci were narrowed. Marked dilatation of the superficial blood vessels was noted. Virtually the entire left frontal lobe had been resected. The right lateral ventricle was collapsed. Residual tumor was evident along the line of surgical excision, with extension into the temporal lobe and the basal ganglia.

Microscopic Examination. There were broad sheets of tumor cells within the subarachnoid space (Fig. 3), and invasion of the adjacent dura mater. The cerebral cortex of the remaining left frontal lobe also showed tumor cells within the perivascular spaces. Focal hemorrhage and necrosis were present, but there was no calcification.

DISCUSSION

Most of the current textbooks of pathology and pediatrics fail to mention the oligodendroglioma as a tumor to be considered in the young-age group. Nelson[6] stated that the cerebral hemispheres of children are rarely invaded by tumor, but if they are, one of the most common types is the malignant sarcoma of the meninges.

Earnest and his co-workers,[4] in a comprehensive survey of 200 cases of oligodendroglioma, found the highest incidence in the fourth and fifth decades of life. No age group, however, was spared, and this particular neoplasm was surprisingly frequent at both extremes of life. The same study included 7 cases in the first decade, and 19 cases in the second decade of life. No appreciable sex difference was found, but the cerebral hemisphere was the primary site affected. In 10 instances other sites, such as the thalamus, lateral ventricle, or region of the optic chiasm were involved.

Shenkin *et al.*[7] found that tumors of the ventricles occurred at a significantly earlier age than those in the cerebral hemispheres. The third ventricle was the site of origin in 4 cases, but no patient in this group was younger than 19 years of age.

Most studies have stressed calcification as one of the important criteria in the histological diagnosis of oligodendroglioma. Martin and Lemmen[5] reviewed a series of 1577 varied intracranial lesions, correlating the incidence of calcification with histological type. They found calcification in every major type of cranial neoplasm except the schwannomas and hemangioblastomas. A total of 34 oligodendrogliomas were included in the study, and slightly over half (18) showed calcification. It is interesting to note that in the latter study 3 oligodendrogliomas were found in the age group 0–15 years. Although no details are recorded, 2 in this group demonstrated calcification.

The immediate cause of death in Case 1 was probably intraventricular hemorrhage, which occurred secondary to necrosis and hemorrhage of the tumor in the third ventricle. The death in Case 2 was related to the massive infiltration of the neoplasm throughout the left frontal lobe.

In the latter case there was evidence of seeding within the subarachnoid and perivascular spaces. There is considerable evidence to support the view that such dissemination is more frequent than generally thought.

Blumenfeld and Gardner[3] found oligodendrogliomas second only to medulloblastomas in tendency to disseminate through the subarachnoid space and ventricles. Earnest *et al.*[4] found evidence of seeding in 14 of 165 cases. Trowbridge and French[8] reported an interesting case with dissemination throughout the fourth ventricle and leptomeninges. The above evidence indicates that subarachnoid seeding and dissemination are not unusual features of these tumors in adults or children.

SUMMARY

Two cases of oligodendroglioma occurring in the young-age group are presented. Some of the current features with respect to subarachnoid seeding and dissemination are discussed briefly.

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STUDIES OF TRIGEMINAL NERVE POTENTIALS

IN NORMAL COMPARED TO ABNORMAL EXPERIMENTAL PREPARATIONS*

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SUCCESSFUL operations for the relief of trigeminal neuralgia were initially developed on the basis of anatomic studies of the trigeminal system.[2,3,5,12] However, current modifications in the surgical treatment of tic douloureux[10,11,13,14,16] suggest that altered physiologic properties of the 5th cranial nerve may be responsible for the clinical successes reported following these modified procedures. Therefore we have undertaken an experimental study of the trigeminal system using electrophysiologic techniques.

Three phases of investigation have been initiated: an electrophysiologic survey of the evoked potentials in the trigeminal system of normal cats and monkeys; the preparation of a chronic syndrome in cats characterized by over-reaction to tactile facial stimulation with an electrophysiologic study of this altered trigeminal system; and a study of trigeminal nerve potentials in patients with and without tic douloureux. If any portion of the results of the first two phases have a bearing on the clinical syndrome of tic douloureux, such a correlation will depend entirely upon the results of the third phase of the study.

Electrophysiologic studies of evoked trigeminal potentials in normal cats have been reported.[1,4,8,9] The present study of the second phase of this problem is based on observations obtained in experiments on 5 normal monkeys and 30 normal cats compared with 17 chronic cat preparations.

METHOD

Several techniques were employed in attempts to produce a laboratory animal with chronic over-reaction to tactile stimulation of the face. Thorotrast* and penicillin in oil injected into the gasserian ganglion occasionally gave mild hypesthesia. Commercial alumina gel[6,7] injected into the main sensory nucleus of V caused no over-reaction to stimulation. Alumina gel injected into the gasserian ganglion caused slight hypesthesia initially which gradually diminished and 6 to 8 months later facial stimulation was associ-

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† Markle Scholar in Medical Science.

* Lestagar and Co., Inc., Detroit, Michigan.

ated with intermittent weak pawing of the operated side in 2 out of 6 animals. It seemed evident that these 2 animals were experiencing a mild abnormal sensation. However only when 0.02 to 0.03 cc. of a weak alumina gel suspension was injected into the spinal nucleus of V, did tactile facial stimulation cause a striking and abnormal response.

The recording system for evoked potentials in these animals is illustrated in Fig. 1. Using a Grass stimulator and isolation transformer single monophasic stimuli of 0.01 msec. duration were applied to the infra-orbital nerve,

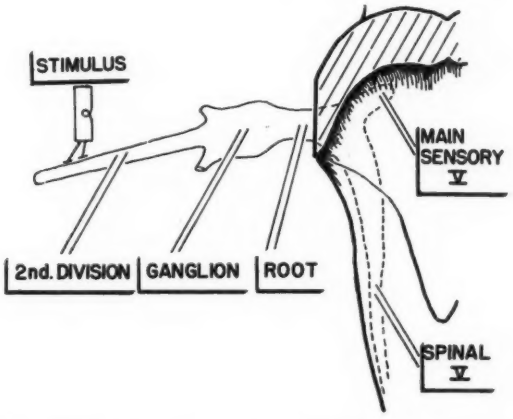

FIG. 1. Stimulating electrodes are placed on the infra-orbital nerve. Bipolar steel recording electrodes are inserted into the 2nd division, gasserian ganglion and posterior root in the middle fossa and the primary sensory trigeminal nuclei in the brain stem.

and increased in voltage from that required to evoke a threshold to that required for a maximum response in the 2nd division of the trigeminal nerve. Bipolar steel electrodes with uninsulated tips measuring 60 to 80 μ in diameter were placed in the 2nd division of the trigeminal nerve, the gasserian ganglion, the main sensory nucleus, and the trigeminal spinal nucleus and tract. A conventional push-pull amplifier with oscilloscope was used. Light Surital* anesthesia, Anectine† and artificial respiration were employed.

RESULTS

Recordings from the 2nd division, gasserian ganglion, and posterior root of a normal cat following stimulation of the maxillary nerve are illustrated in Fig. 2. The afferent potential (a) immediately follows the stimulus artifact. A delayed potential (b) can also be seen, lasting approximately 10 msec. It is activated at thresholds corresponding to touch fibers and has received particular attention in the course of these and earlier studies.[12]

In Fig. 3 (monkey) the potentials were recorded in the 2nd division in

* Parke-Davis and Co., Detroit, Michigan.
† Burroughs Wellcome and Co. (U.S.A.) Inc., Tuckahoe, N.Y.

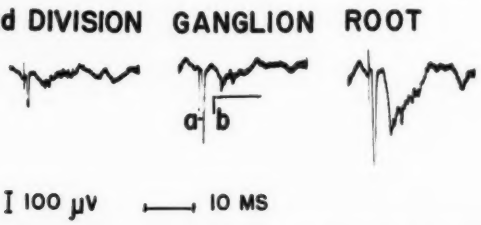

FIG. 2. Recordings following stimulation of the infra-orbital nerve of a normal cat, showing maximum responses of the evoked trigeminal potentials from the 2nd division, gasserian ganglion and posterior root. The evoked afferent spike potential (a) is followed by a delayed potential (b) which is conducted centrifugally and lasts approximately 10 msec.

the middle fossa and spinal V. The afferent spike can be clearly seen (a) with a delayed potential following it (b). In spinal V the afferent spike is again illustrated (a) with delayed activity (b). A tractotomy was performed in this animal at a level 2 mm. above the obex of the 4th ventricle and 2.0 mm. caudal to the spinal V recording electrode. Following the tractotomy, although the afferent spike (a) is still clearly evident, the delayed activity is markedly reduced or absent. Posterior root section also abolishes this delayed activity in the peripheral nerve. These two features indicate centrifugal conduction of the delayed activity into peripheral components of the trigeminal nerve from a source between the lower medulla and the upper cervical region.

After the evident source of this activity was identified within the caudal portion of the spinal trigeminal system, the second phase of the study was initiated.

An alumina gel suspension was injected into the caudal portion of spinal V in cats. The majority showed no immediate after-effect. Some vestibular imbalance was noted in 3 preparations. One to 2 months later they began to lose hair from the eyebrow and the upper lip homolateral to the side of injection. The cleansing ritual was neglected and thin incrustations accumu-

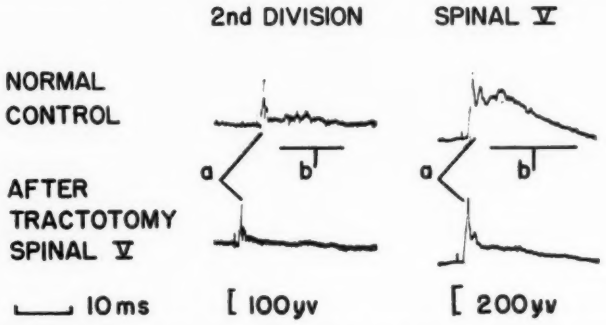

FIG. 3. Recordings (normal monkey) from the 2nd division of the trigeminal nerve and spinal V show a fast afferent spike (a) and delayed activity (b). The delayed activity is markedly reduced or abolished following a tractotomy of spinal V caudal to the spinal V recording electrode.

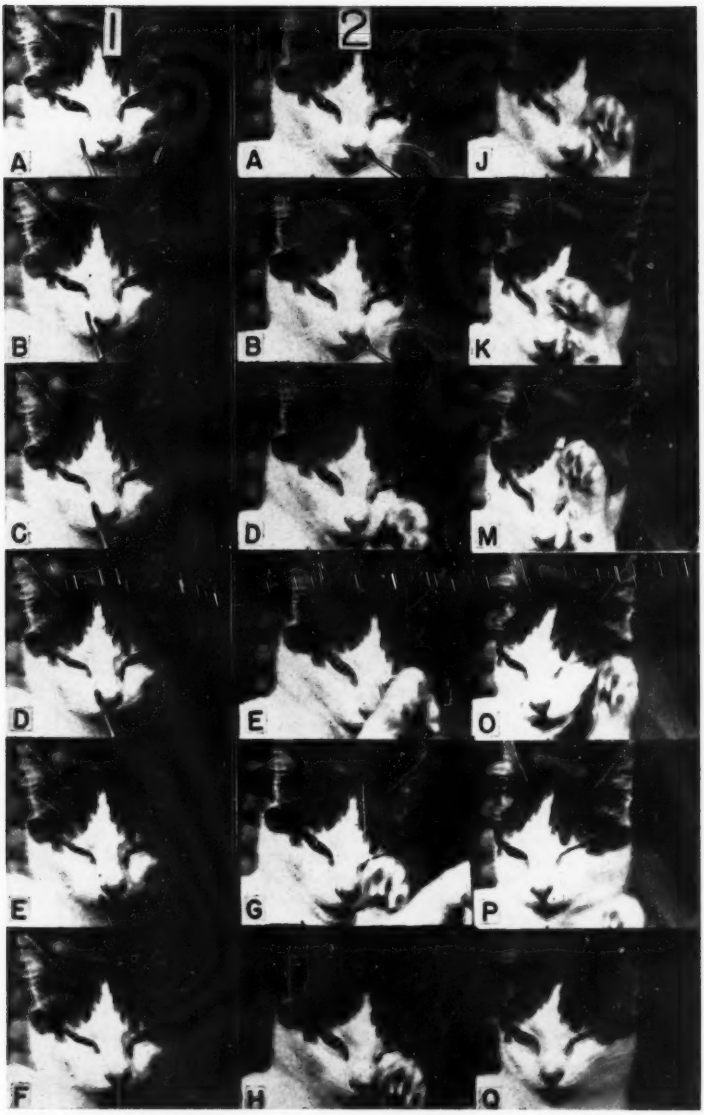

FIG. 4. A dilute suspension (0.03 cc.) of commercial alumina gel was injected on the left side into the caudal portion of spinal V below the obex of the 4th ventricle 4 months before these pictures were made. Series 1 indicates the normal response following light tactile stimulation on the right. Series 2 indicates the brisk onset and prolonged over-reaction which follows similar stimulation on the left. Consecutive frame letters indicate a deletion of every second frame from the original movie film. The interruption of consecutive lettering in Series 2 indicates further deletions between 2-B and 2-O.

lated on the inner canthus and nose of that side. The corneal reflexes remained equal.

Approximately 3 months following injection, these animals developed marked over-reaction to tactile stimulation on the side of the face corresponding to the side of the alumina gel injection (Fig. 4:2). Trigger points could be demonstrated in that the blunt end of a pin touched very lightly to a few hairs at the inner canthus or lateral to the nares on that side caused a brisk squint, twitch and withdrawal. Frequently the animals pawed at

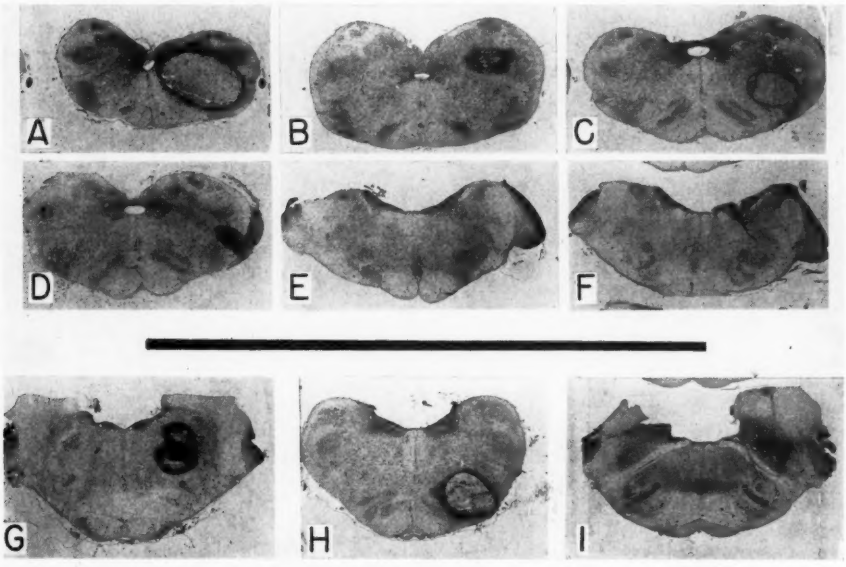

FIG. 5. Alumina gel lesions in the medulla of 6 chronic over-reactive cats are illustrated in sections A–F. Sections, G, H and I are 3 of a series of control lesions. Sections G and I are sections in the region of the main sensory nucleus and in section H the lesion is ventral to spinal V. Each illustration has been chosen from the portion with the largest diameter. Sections A–F were made 4 to 5 months after the injections were made. The control sections were examined after an 8- to 12-month period of observation.

the air on the affected side. Such a response could also be elicited occasionally from the 3rd division and from the anterior portion of the external ear, although never from the posterior two-thirds of the ear. Two apparently spontaneous paroxysms of this character have been observed. No such response was elicited on the uninjected side (Fig. 4:1).

Ultimately the animals avoided all contacts with the affected side of the face, and would not eat. Warm milk brought in contact with the whiskers was vigorously avoided. At this point they were maintained on parenteral fluids and tube feedings to prevent rapid deterioration.

Sections A–F of Fig. 5 illustrate lesions from 6 such chronic animals. In each instance the sections have been chosen from the region of maximum diameter of the lesion. In each, the lesion involved the nucleus of spinal V.

No animal to date with a verified injection of alumina gel into spinal V (that has lived 3 months) has failed to show this abnormal response. Sections G, H, and I represent 3 of a series of control preparations. The control preparations G and I demonstrate alumina gel lesions in the region of the main sensory nucleus. In Section H, the alumina gel was placed ventral to spinal V. None of these preparations developed any abnormal response to tactile stimulation.

Evoked potentials from the 2nd division of the trigeminal nerve in the middle fossa in 3 normal cats are shown at the top of Fig. 6. Recordings in

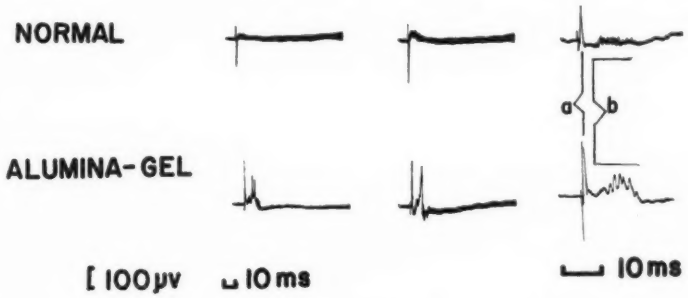

FIG. 6. Recordings from the 2nd division of the trigeminal nerve in the middle fossa following stimulation of the infra-orbital nerve. The four on the left were obtained at a slower time line than the two on the right. Three normal records are illustrated above and 3 from chronic preparations with marked over-reaction to tactile facial stimulation are shown below. That in the right-hand lower corner was recorded from the preparation shown in Fig. 4. There is a clear increase in the amplitude of the delayed activity (b) in the lower records compared to the control records above.

the lower line are from 3 cats in which alumina gel had been injected into spinal V. Each of these 3 preparations demonstrated over-reaction to tactile facial stimulation (as in Fig. 4:2). The delayed potential varies from 60 to 80 per cent of the amplitude of the afferent spike in the experimental chronic animals in contrast to 10 to 25 per cent of that size in the normal animals.

From a clinical standpoint it is of some interest that in a chronic animal with violent over-reaction to tactile stimulation, mild contusion of the gasserian gangion was followed by a normal response to tactile facial stimulation without demonstrable hypesthesia. At the end of a 2-week observation period histologic examination confirmed the location of the alumina gel in spinal V and showed mild chromatolysis and round cell infiltration of the gasserian ganglion.

In other chronic preparations it has been a common observation that mild trauma to the ganglion caused by repeated electrode placement will markedly reduce or abolish entirely the unusually high voltage delayed activity.

DISCUSSION

These animals appear to experience an unpleasant sensation following tactile stimulation. The interpretation of this behavior must be guarded.

Injection of alumina gel into the main sensory nucleus, which is the primary touch fiber relay station, fails to produce this reaction. Injection into spinal V, on the other hand, consistently produces this response, despite the very limited representation of touch in this center.[15]

In these apparently oversensitive animals, there is a 300 to 600 per cent increase in the recorded voltage of the centrifugally conducted delayed activity in the peripheral divisions of the trigeminal nerve. The source of this activity is in the region of the subnucleus caudalis of spinal V and although it has been seen to increase in amplitude with greater stimulation, it is first activated at the same threshold as are touch fibers in the peripheral trigeminal nerve.[8] It may be that under these circumstances the increased voltage of the delayed activity in the peripheral trigeminal nerve and the over-reaction to tactile facial stimulation reflect increased activity at the central terminus of those touch fibers which do synapse in spinal V.

As the primary pain relays for the face are known to be centered in spinal V,[12] it is possible that components of the touch and pain modality mechanisms have been altered in these chronic preparations so as to allow induction of a *painful* sensation by *tactile* stimulation.[8] Under abnormal conditions of increased neural activity in the nucleus caudalis then, the larger touch fibers may transmit delayed potentials of higher voltage as direct evidence of the increased neural activity at the source of the delayed potential.

Under these circumstances, with over-reaction to tactile facial stimulation and increased voltage in the delayed activity, a direct correlation between the two phenomena may not be justified. The change in the delayed activity in the peripheral nerve may not relate primarily to the animals' altered behavior. The increased neural activity in spinal V may relay to higher centers in the thalamus or even cortex and the over-reaction to tactile stimulation may require that the secondary cephalic relay be intact.

Preliminary recordings of thalamic potentials however have shown no difference in amplitude or duration of single evoked thalamic responses in the acute compared with a limited number of chronic over-reactive preparations. Decerebration at the superior colliculi has not altered the delayed potential in the peripheral nerve in acute preparations but has on the other hand abolished the over-reaction to facial stimulation. This phase of the study is under investigation at the present time using modifications of the techniques described in this report and acute rather than chronic over-reactive preparations.

An altered neural mechanism in spinal V alone does not seem adequate to explain all of our observations. Mild trauma to the trigeminal nerve in the region of the gasserian ganglion markedly reduces or abolishes the unusually high voltage delayed activity in chronic preparations and can eliminate the overreaction to tactile facial stimulation in these cats. After such trauma, there is no gross evidence of hypesthesia to account for the decrease in sensitivity. Furthermore, the threshold for an afferent evoked potential remains unchanged in the same division of the trigeminal in which the de-

layed activity is no longer apparent. It may be, therefore, that the delayed activity that is conducted centrifugally from spinal V alters the state of excitability of cutaneous end organs and thereby alters the normal response to tactile stimulation through its influence at the sensory end organ. Such an influence at present must remain conjectural.

SUMMARY

1) We have presented some observations made during electrophysiological studies of the trigeminal system in normal cats and monkeys.

2) An afferent spike recorded in the peripheral trigeminal component is followed by a delayed potential which arises from spinal V between the first cervical segment and the lower medulla. A portion at least of this delayed activity is activated at touch thresholds and is conducted in a centrifugal direction from the brain stem back into the peripheral elements of the trigeminal nerve.

3) Over-reaction to tactile stimulation of the face has been produced in chronic preparations by injection of alumina gel into the caudal portion of the spinal tract and nucleus of the trigeminal nerve. In these animals a marked increase in voltage has been noted in the centrifugally conducted delayed potentials in the trigeminal nerve.

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SOME PRACTICAL ASPECTS IN THE REHABILITATION OF PARAPLEGICS*

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I CAN'T tell you how glad I am to be back in circulation again. As I pick up the segments of neurosurgical knowledge that have grown into accepted ideas during my absence, I realize what a long time it has been. This feeling makes me hesitant about opening my mouth at all. I am apprehensive that I may turn out to be some sort of Rip Van Winkle; or, even more horrible, a slightly nauseous subjective raconteur.

My gratitude to the many people along the line who have helped me is more than I can possibly express. This has included not only elaborate Veterans Administration programs but the intimate help and advice of a number of individuals including several of yourselves. Without it I could have gotten nowhere. Nowhere, somewhere—I am not at all sure that I am getting "somewhere," but I do know it is very important to me that I think so. As a matter of fact, as I recall it, this type of self-indulgence is fairly essential to even a normal neurosurgeon.

There are certain impressions still vivid for me that may have some significance and it is one portion of these that I wish to pass on to you today. I want to talk to you in a critical and I hope constructive way about some of the aspects of the popularly accepted rehabilitation program for paraplegics.

It is absolutely essential that each paraplegic at the right time be taught the tricks of getting himself around and the fundamentals of bladder and bowel training. So paraplegics are of necessity being rehabilitated in hospitals instead of homes. The difference is vast and the ultimate transition must be wisely anticipated. Take crutch-walking for example. Many units lay great stress on the importance of crutch-walking. There can be no question as to the physical and metabolic arguments behind this policy, but it seems to me that this emphasis is not only disproportionate to the value derived but that it is often detrimental. It is now common practice to get a patient going on body-building exercises and crutch-walking while his wound is still fresh or the disease process is still active. I am not at all sure that this is wise, particularly when it is promoted at the feverish clip now popular with physical

* Presented at the annual meeting of the American Academy of Neurological Surgery, Hot Springs, Virginia, October 28, 1955.

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medicine chiefs and their therapists. The eager and cooperative patient is likely to end up in a do-or-die frame of mind not unlike that of a Sophomore trying to make the Varsity; and all of this over the rapid acquisition of a fiendishly grotesque gait of quite doubtful value.

The danger of this philosophy runs even deeper. Time, energy, and funds are expended so lavishly that pretty soon both physician and patient get the idea that fancy crutch-walking is the proud evidence of successful rehabilitation. The chief shows off his prize performers to visiting doctors who are lavish in their praise; but the truth of the matter is that unless the average paraplegic's cord lesion is quite low and his determination unusually high, his crutch-walking won't earn him many dollars. And the earning of dollars is much closer to true rehabilitation than any amount of crutch-walking. Sensing your raised eyebrows, I will return to a defense of this statement later. As I watched the ebb and flow of patients in the particular center where I received invaluable rehabilitation experience, I could not but be impressed by the fact that, although many were discharged on crutches, all but a few returned in wheel chairs. It was the rare returning "graduate" who was earning a substantial part of his living from crutches. The vast majority who were partially self-supporting were working from wheel chairs; and yet all of these had received intensive crutch-training. For most of those who work, it just isn't practical. The energy required for crutch-walking is too great and the penalties of a bad fall are too severe. If I should attempt to walk from my parked car to my office, it would be about the equivalent of a vigorous squash game; while a broken wrist would mean total loss of hard-won independence and earning capacity. At one stage I worked intensively at crutch-walking and became fairly adept at the wretched gait. I wish I were better at it and maybe I some day will be. But right now my wisest use of it seems to be only for exercise, much as a normal person might take time out for a game of tennis. To work I must use a wheel chair.

These are practical considerations for the *average* paraplegic, not the star performer. The whole story could be repeated with bladder training. I am sure you will see the parallel; suffice it to say that any bladder-training program which leaves a man with uncontrolled stress incontinence is a practical disappointment if not failure no matter how great the academic success implied by the term "automatic bladder." Stress incontinence is no joke. Unless there are means taken to recognize it and meet it, the patient is incapacitated for social or vocational freedom. Here again there is the great difference between hospital and home rehabilitation. It is one thing to be able to press a button and have complete bedding or clothing changes provided as part of a hospital system; it is quite another to confront a wife or mother with this problem. It is one thing for a paraplegic to soil himself while watching television among other paraplegics on a ward; it is quite another thing for him to encounter this same hazard in the outside world while in a machine shop or at a cocktail party. There is a tendency in American bladder-training programs to discourage the use of stress incontinence urinals on the grounds that they lead to the loss of good habits. Some of this is warranted; but some

of it also is unrealistic if rehabilitation at work and at play among normal people is the true object of the program.

In both of these concepts, bladder- and crutch-training, the Canadians are more practical than ourselves. The group working in Toronto would, I think, be in pretty close accord with much of what I have said. The emphasis in the Canadian rehabilitation program is on getting the patient back into his normal environment and, with this in view, a more practical attitude toward crutch- and bladder-training is emphasized.

I would like to talk with you for a few minutes now about some of the paraplegic preoccupations which can become just as disabling in their way as the intrinsic organic lesion. It is to man's credit that, faced with severe physical adversity, he usually does surprisingly well. The so-called optimism and courage of a well-adjusted paraplegic is less conscious than instinctive. You yourself would have it under similar circumstances. It seems almost as if this endearing instinct of the human race remains hidden until the stimulus of hardship brings it out in any one of us. It forms the outer shell behind which a handicapped person lives. Part of this facade is bluff, a phenomenon differing only in degree from its manifestation in normal life. The paraplegic simply must have more of it; for the preoccupations which are bound to possess him if the bluff fails are almost all disturbing and unpleasant. Here are a few of them.

First of all, sensory discomfort. The term "paresthesia" covers a realm of discomfort about which the average neurologist who has not experienced it can have only the dimmest academic suspicions. In fact, I suspect there are neurologists who don't even really know the meaning of the word "anesthesia" as experienced by a patient. True anesthesia, meaning lack of sensation, is practically non-existent in a paraplegic. He has so many sensations, all of them unpleasant, that if suddenly experienced by an otherwise normal person they would be sufficient to send such a one screaming for emergency help. I have often discussed these sensations with fellow patients. They follow the most bizarre patterns and seem to encompass all sensory modalities. One man feels as though his naked legs were hanging out of a window on a freezing cold day. Another describes sky rockets bursting in the soles of his feet; while still another feels a family of snakes squirming in his buttocks. Others constantly and deceptively experience the urgent agonies of a full bladder. These are but a sampling, for each paraplegic's sensations are very much his own.

Then there is the preoccupation with social insufficiency and its companion, financial insecurity. The paraplegic father or mother, husband or wife, finds it hard to forget that he is only a poor substitute for what he might have been. This is exaggerated by the difficulties he encounters when he tries to become a social unit, whether it be simply going to a party at the home of a friend or encountering the more complicated contacts involved in travel. Moreover, there is the inescapable economic fact that the paraplegic is probably not earning as much as he would have normally in support of himself or his family.

These are the types of preoccupation ready and willing to overwhelm a paraplegic. And yet, unlike the paralysis, these factors are subject to modification. They can be made worse and they can be made better. They can be exaggerated or they can be minimized. Certainly there are many moments in the life of a paraplegic in which he completely forgets that he is uncomfortable or socially insufficient. It may be helpful to analyze how these moments can be brought about because it is in them that the paraplegic becomes a useful and self-respecting member of society.

They are most reliably brought about by intense *enthusiasm*. It makes little difference whether that enthusiasm be for a beautiful girl, a fascinating piece of writing, an exciting ball game, or an absorbing piece of work. The result is the same. The paraplegic under its spell may become totally unaware of his paresthesia, his social insufficiencies, or financial problems.

If enthusiasm is the most important therapeutic agent for the sublimation of detrimental preoccupation, the continuing acquisition of *new increments of social independence* runs a close second. Although the wise paraplegic indulges no thought of miraculous recovery, he is constantly searching for the new trick or circumstance that will increase his sense of social independence.

Since these two factors are so important to the paraplegic, what can be done in a tangible way to invoke their indispensable aid? Here are some things that can be done. A highly individualized search can be made for those interests that bring about an enthusiastic response. Much of this responsibility is now handed over to departments of occupational therapy who do a tremendous job with it. I suspect that no one, however, is better suited to find out the real sources of enthusiasm that fit a given paraplegic's life than his own sympathetic physician or surgeon. The paraplegic will not need much of a start; once the enthusiasm is discovered and tapped, he will develop it eagerly. But particularly in the age group in which paraplegia is common, namely the immature 20's, the average individual often has not discovered his own enthusiasms and needs the mature help of his physician to do so. And so I suggest that part of your job in the care of a paraplegic is to help him make this discovery about himself, not solely by filling out a requisition for occupational therapy but by the exercise of your own human sympathies and understanding. You may have forgotten, as I had, how glamorous the title of neurosurgeon still is to a patient and how influential you can be as a confidant just because of this title and all it stands for.

A second means of releasing this therapeutic supply of enthusiasm is, I believe, contact with normal people. In spite of the physical and spiritual protection afforded by the isolation of a paraplegic ward, the paraplegic is a happier and more self-respecting individual in his own home or at a party surrounded by normal friends. I am not at all sure that paraplegics are good for each other and I think if I were designing a new hospital ward for their care I would explore very carefully the ways and means of having them mixed with interesting people who are not paralyzed. For many purposes this would be impractical, but the value of the therapy should be recognized

wherever it can be utilized. This does not necessarily mean importing a group of debutantes to swish about among the paraplegics for an hour every Thursday evening. It does mean finding out when the paraplegic's own normal friends are having a party and then seeing to it that he gets there. In such a group he very quickly will assume his old role, whatever it was, and will be taken for what he genuinely is, which is all any one, paraplegic or normal, really asks. The same concept can be applied to employment. All things being equal, I think a paraplegic who is the only one working from a wheel chair in a given assembly line or in a given office will come much closer to his maximum performance as an individual than he would if he were one of eight or ten wheel-chair patients lined up on the same assembly line or in the same office.

So much for the release of enthusiasm. How can we increase the paraplegic's sense of social independence? Many of the ways in which this can be done are simply the product of lively imagination applied to a particular individual's needs. In this sense every smooth-running home of a paraplegic can provide suggestions for the novice. Thus it might be profitable for a new paraplegic just leaving the hospital to be taken on a tour of two or three homes in which other paraplegics have been successfully acclimated. The paraplegic would see things which might escape the eye of a less intimately interested person. Or it might be worth while to compile in a single book or pamphlet two or three hundred of the original devices created and used by paraplegics and distribute this booklet to all it might help. But in this realm of thought, I would particularly like to emphasize the acquisition by the paraplegic of the ability to drive his own car. I doubt if there is a single element in his whole rehabilitation that would contribute so much to his self-respect as the discovery that he can once more travel about his business and about the country with dependence upon practically nobody. Where this idea has been well established there is a carefully planned car-driving training program. After passing an appropriately stiff examination by the State Motor Vehicle Department, the paraplegic is licensed to operate a hand-controlled vehicle. The hand controls can be purchased as a unit which costs in the vicinity of $250 installed on any car. Thus, if the paraplegic can afford a car at all, the additional expense is not a serious barrier. Once he is in his car he is in many ways back in the world again. And of course the values are much more specific than just what it does for his morale. The operation of his own car may be the most significant factor in getting him back to remunerative work.

A final consideration in this search for favorable modification of paraplegic preoccupations is the importance of the well-earned dollar. When I said earlier that I felt the earning of dollars was closer to true rehabilitation than any amount of crutch-walking, I meant just that. Since money is probably here to stay, there is nothing quite so consoling to a paraplegic as the thought that in spite of everything he is consistently able to earn a certain amount in exchange for an honest contribution to society. This honestly earned pay check means not only a new dress for his wife but a growing

respect for himself that he was able to provide it. It is part of our job in the care of paraplegics to make every effort to see that they are fitted for some kind of work which brings them the satisfaction of a well-earned dollar. That is why I say that any portion of the physical rehabilitation program that jeopardizes the adult paraplegic's earning capacity can very quickly become more of a liability than an asset.

Any consideration of the financial burden facing the paraplegic would be incomplete if it did not call to your attention the inadequate means available for the care of civilian paraplegics in the United States. The highly developed Veterans Administration program reaches only a fraction of the total paraplegics in the country. The few private rehabilitation units available to the civilian are still very expensive. Thus the average person trying to bear the burden of rehabilitation for himself or a dependent is faced by an almost insurmountable financial obstacle.

At present in this country we are following the policy that civilian paraplegics should be rehabilitated as part of the general rehabilitation program. Thus they will benefit from the Vocational Rehabilitation Act passed by Congress in 1954. Under its terms the Federal Government is prepared to assist state programs in many ways and to a degree not generally realized. The National Paraplegic Foundation, which represents the paraplegics as well as acts as a fund-raising organization, is developing a steadily growing program in support of related research. Isolated private rehabilitation units such as those in Boston and New York City are doing an outstanding job for the limited groups they serve, and are steadily increasing in number.

There is considerable disagreement in this country as to the advantages of a coordinated program in behalf of paraplegics, such as is maintained in Canada where significant private, state, and federal funds are mobilized for the treatment and rehabilitation of all paraplegics regardless of age, sex or occupation. It seems to me that the ultimate solution to our problem calls for a similar organization coordinating the sincere intentions of the National Paraplegic Foundation and the funds of the federal Vocational Rehabilitation Act, plus the individual, state and industrial programs, into one over-all plan for the acute and chronic care, as well as the vocational rehabilitation, of all paralyzed individuals.

SUMMARY

Thus in the few moments available I have discussed some of the ways in which we can be more practical in our attitude toward the rehabilitation of paraplegics. Some dangers of an overenthusiastic physical-training program have been pointed out. The importance of releasing enthusiasm and a sense of social independence as specific therapeutic agents in the modification of harmful paraplegic preoccupations has been indicated, and the importance of economic independence in the reestablishment and maintenance of the paraplegic's self-respect has been emphasized.

ANASTOMOSES BETWEEN CERVICAL NERVE ROOTS*

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THE distribution of subjective pain and objective sensory disturbance caused by cervical root involvement frequently has been noted to deviate from the "normal" dermatome pattern. Although variation from the normal may be explained on the basis of pre- or post-fixation of the brachial plexus and by peripheral overlap, recent experience has led us to consider another possibility.

In the course of performing posterior rhizotomy for relief of limited pain in the hand we have had a patient in whom slender anastomotic branches were found running from the dural margin of the 7th posterior cervical root to join the 6th cervical root centrally. Under local anesthesia, stimulation of the anastomotic rootlet produced discrete pain in the thumb and thenar eminence. Stimulation of the 7th root, which this branch accompanied peripherally, produced pain in the index finger. In another patient, with pain in the ulnar aspect of the hand, an anastomotic ramus from C8 to T1 was found and had to be cut along with the C8 rootlets to effectively abolish pain.

In two patients with cervical disc herniation, discrepancy between sclerotome and dermatome levels could not be explained on the basis of anomalous fixation of the plexus. Intradural exploration revealed compression of a rootlet running centrally to join the posterior root at the next segment. Similar anastomoses of the upper cervical nerves have been found frequently in the course of operations for high cervical cordotomy.

These experiences therefore have led us to consider the role of intrathecal anastomoses between the cervical posterior roots as an additional factor to explain variations from "normal" dermatome patterns. Observations made in anatomical dissections will be presented.

OBSERVATIONS

The cervical spines of 13 adult human subjects were dissected and the findings are diagrammatically represented in Fig. 1. Anastomotic rami from the peripheral portion of one sensory root to the central portion of an adjacent sensory root were found in all cases. Frequency varied from a solitary connection between C6 and C5 on one side in one subject, to a maximum

* Presented at a meeting of the American Academy of Neurological Surgery, Hot Springs, Virginia, October 29, 1955.

of six anastomotic rami involving various roots on both sides in two subjects. Of the 12 subjects with multiple anastomoses, approximate bilateral symmetry was noted in only one, with branches from C7 to C6, and T1 to C8 on both sides; in this case, additional rami were found between C5 and C4 on the left and C4 and C3 on the right.

The most frequent communications in this group of dissections were between C7 and C6. It was noted that the majority of anastomoses appeared to arise from the ganglion of the nerve below to enter the cord with the posterior root fibres of the next rostral nerve. Far less commonly did the branch enter with the root below. None of the anastomoses in these specimens extended farther than one segment; however, we have found one connection between C6 and C4 at operation in one patient.

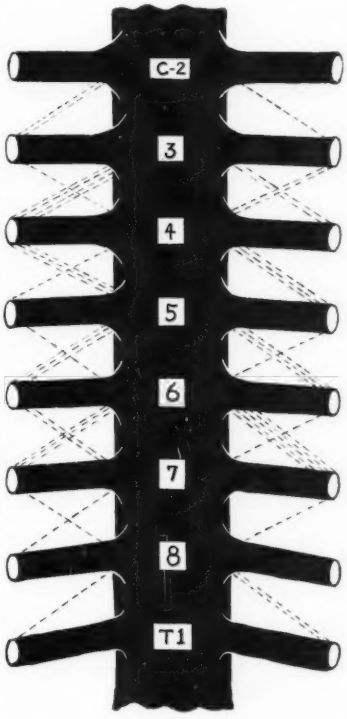

FIG. 1. Intrathecal anastomotic branches of posterior rootlets found in 13 dissections of cervical cord. Dotted lines are oriented in direction of anastomoses from parent ganglion laterally, coursing medially to join neighboring rootlets entering the cord.

DISCUSSION

The anatomic observations made in this small series of cadaver dissections indicate that intrathecal anastomotic connections between the posterior roots occur quite commonly in the cervical region. Since our dissections were confined to the neck, it cannot be said whether or not they occur similarly in the lower portions of the spinal axis. The ground structure exists embryologically to permit such anastomoses to occur throughout the spine. In the human embryo, Streeter[2] observed that, by the 20th day, the ganglion crest of the hindbrain and spinal cord divides longitudinally into right and left halves. Projecting ventralward from the dorsal bridge are the primitive spinal ganglia (Fig. 2A). At 4 weeks, the dorsal border of the spinal crest still forms a continuous bridge along the tops of the primitive ganglia throughout the length of the spinal cord (Fig. 2B). The dorsal roots are much slower to develop than are the ventral roots, and the ganglionic masses do not begin to separate until about 30 days. The interganglionic ridge disappears at the end of the 5th week (Fig. 2C). At this time, as Streeter puts it, "one gets the impression of an actual conversion of the dorsal bridge into the ganglion rootlets." These rootlets increase in length "and show a tendency toward anastomosis." Streeter's reconstruction of a 6-week human embryo shows anastomosis between C3 and C4 dorsal roots (Fig. 2D).

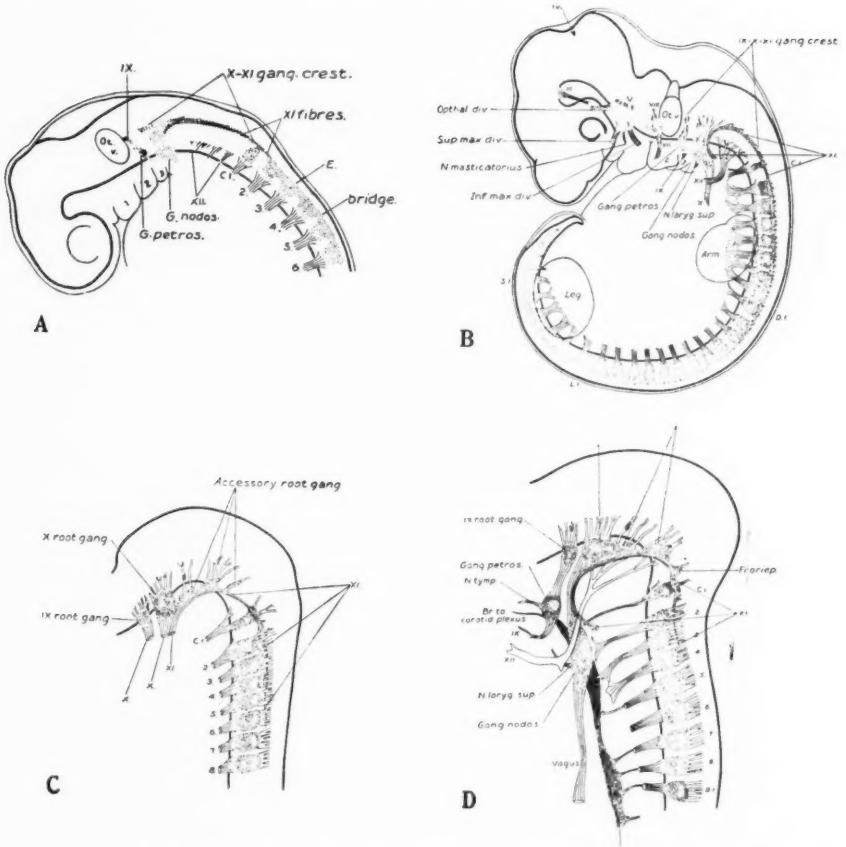

FIG. 2. Human embryos. (A) 20 days. (B) 4 weeks. (C) 5 weeks. (D) 6 weeks. The interganglionic bridge begins to disappear at 5 weeks. In D, anastomosis between the dorsal rootlets of C3 and C4 is illustrated. (Modified from Streeter.[2])

If it should be true that dorsal root anastomoses occur more often in the cervical region than in the more caudal areas, then we have to look for some mechanism in addition to the presence of the interganglionic bridge which extends all the way down the spinal axis. Kazzander,[1] concerned with the relationships of the spinal accessory nerve, found that, in dissections of human subjects, when a separate first cervical root existed, the majority had an anastomosis with the accessory nerve. Weigner[3] in 1901, pursuing the same problem of accessory-cervical nerve relationship, noted similar close connections. In addition, he casually noted that "between the dorsal roots of the cervical nerves there exist many different types of anastomoses" and that the arrangement on the two sides is not always similar. Examples of some of the anastomoses found by Weigner in 37 dissections are shown in

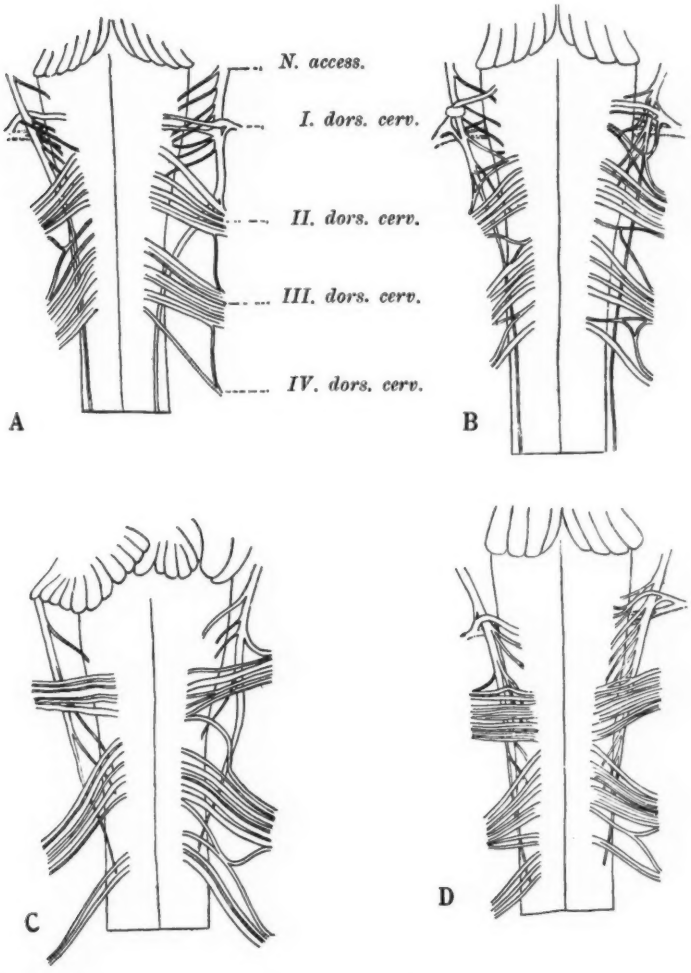

FIG. 3. Examples of anastomoses of dorsal roots in the upper cervical region associated with the spinal accessory nerves. (Modified from Weigner.[3])

Fig. 3. Weigner noted that the accessory usually arises from the level of C4, but may begin a segment higher or lower. On the basis of Weigner's observations and his own studies, Streeter postulated that the accessory nerve, being laid down earlier, may guide the impinging dorsal rootlets along its own trunk. If this is correct, then we have a reasonable embryological explanation for the common occurrence of dorsal root anastomoses in the cervical region.

From the clinical point of view, the occurrence of intrathecal dorsal root

anastomoses gives us another explanation for variations from the "normal" dermatome pattern of pain distribution and sensory impairment. When we have found these connections at the time of operation, we have noticed that the anastomotic branch courses close to the dura mater as it leaves its parent ganglion and may be overlooked in performing posterior rhizotomy, unless the entire root is exposed between the cord entry zone and dural sheath. The omission of section of such a branch may at times account for failure of rhizotomy to relieve pain.

SUMMARY

Cadaver dissections and observations in the living patient have demonstrated the presence of intrathecal anastomoses between neighboring posterior nerve roots in the cervical region.

The occurrence of such connections can be explained on embryological factors, including the presence of interganglionic bridges and the developmental relationship between the accessory and the upper spinal nerves.

The presence of intrathecal anastomoses may explain some of the deviations from the "normal" dermatome pattern so far as distribution of pain and sensory impairment are concerned. In performance of posterior rhizotomy for relief of pain, search should be made for the presence of anastomotic branches.

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CASE REPORTS AND TECHNICAL NOTE

FEMORAL NERVE REPAIR*

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There are very few cases of femoral nerve injury in the literature. There are even fewer cases reported in which the femoral nerve has been repaired. The object of this paper is to discuss the problem and to give 2 examples of gratifying repair of this nerve in the pelvis.

The femoral nerve, sometimes called the anterior crural, arises from the lumbosacral plexus. It has its origin in the branches of the 2nd, 3rd and 4th lumbar anterior primary divisions in the substance of the psoas muscle. It curves down over the iliacus and the wing of the ilium just lateral to the femoral artery. It passes under the inguinal ligament and into Scarpa's triangle in the thigh where it divides into cutaneous and motor branches. There it supplies the sartorius and quadriceps muscles and innervates the skin of the anteromedial aspect of the lower extremity.

In World War I, Pollock[2] reported 19 cases of femoral nerve injuries, of which none was repaired. There were undoubtedly a greater number of femoral nerve injuries but because of the proximity of the artery and nerve in the pelvis, most missile wounds resulted in death from hemorrhage. Death from hemorrhage may also occur from a wound of the groin, but a tourniquet or direct pressure may save the individual fortunate enough to be treated promptly.

There were only 21 (0.3 per cent) femoral nerve cases among the 7,050 neurorrhaphies recorded by the Peripheral Nerve Registry in World War II. There have been no reports of any of these being repaired in the pelvis, the majority being in the groin. Nineteen of these were primary repairs; only 2 were secondary.[6] Seletz[4] reported 41 femoral nerve injuries out of 2,037. Many of these were inoperable and a few required only a neurolysis.[5] Grantham and Pollard[1] reported no cases of femoral nerve repair in 281 nerve sutures. The Armed Forces Institute Film Library has an excellent motion picture of a femoral nerve repair made in 1947. This repair was performed in the groin.

Since no detailed report of a femoral neurorrhaphy in the pelvis could be found in the literature, it was felt worth while to report the case encountered at this hospital. In discussing the matter with a colleague, Dr. Albert Starr, he recalled an extraordinary case of Dr. William B. Scoville in which the femoral nerve was repaired in the pelvis. Dr. Scoville[3] was kind enough to send us the case history for inclusion with our own.

CASE REPORTS

Case 1. J.M.B., a 25-year-old seaman, was shot in the abdomen by a .45 caliber automatic slug at short range on Feb. 20, 1952. The bullet entered the abdomen 3 cm. below and

* The opinions or assertions contained herein are those of the author and are not to be construed as official or reflecting the views of the Navy Department or of the naval service at large.

just to the left of the umbilicus. The wound of exit was in the right gluteal area laterally. He noted sudden severe pain and numbness in the leg, inability to move the right leg, and rather severe abdominal pain.

1st Operation. He was immediately taken to the U. S. Naval Hospital, Portsmouth, Virginia, given blood transfusions, and, through a right lower rectus incision, his peritoneal cavity was explored. There were numerous holes in the ileum and jejunum as well as a through-and-through hole in the cecum. These perforations were sutured; a 6-inch section of small bowel was removed and anastomosis was performed. No attempt to explore the femoral nerve was carried out nor was the femoral artery explored since no large hematoma was present.

Course. Convalescence was uneventful. Roentgenograms showed a perforating wound in the wing of the right ilium. Clinically, the patient exhibited a complete sensory and motor femoral nerve paralysis. He had no movement of the iliacus, sartorius nor quadriceps muscles.

2nd Operation. One month later, under spinal anesthesia, a 5-inch oblique incision was made just above Poupart's ligament in the right lower quadrant. The peritoneum was reflected medially and the bullet wound was found penetrating the iliacus muscle and entering the wing of the ilium. There was considerable scarring in this area. On tracing down the femoral artery, a small slightly contused area of the vessel was found. This was so slight that the arterial wall was not materially weakened. Immediately lateral to this the femoral nerve was uncovered and found to be totally severed. Proximally, a central neuroma was present. The nerve was divided with a sharp razor until intact fascicles were encountered. It was then necessary to flex the thigh in order to relieve the tension and to place one permanent #005 tantalum sling suture through the nerve. Fine epineural tantalum #003 sutures were then used. The patient was placed in a body cast with the thigh flexed.

Course. The cast was removed in 3 weeks and he was allowed to extend his thigh gradually.

Five months later there was some regeneration of the femoral nerve. Tinel's sign was present to 4 inches above the knee and crude sensation above this area was present (Fig. 1).

FIG. 1. Photograph showing area of diminished sensation to light touch. Pain and temperature sense were absent in an area measuring approximately 1½" less in diameter. The operative scars are visible.

There was, however, $2\frac{1}{2}$ inches of atrophy of the right thigh compared with the left, and there was no evidence of motor function. The patient was able to walk by leaning slightly forward and permitting gravity to pull his leg forward into position, but was unable to go up and down stairs. He complained that his knee gave out on him if he forgot to use this posture in walking.

The patient was discharged from the Navy. Examination 1 year later showed almost complete recovery of femoral nerve function. Sensation had returned to normal except for the lower 2 or 3 inches of the anteromedial aspect of the lower leg. There was no longer measurable atrophy of the right thigh. The quadriceps muscles were strong and the patient could forcibly extend leg at the knee equally well in both lower extremities. Subjectively, the only complaint registered was the fact that the right leg tired more easily than the left on climbing stairs or ladders.

A letter received in December, 1954, over $2\frac{1}{2}$ years from the time of repair, revealed the patient to be having no difficulties with his leg and he considered it well.

Case 2 (Dr. William Beecher Scoville's case). An 8-year-old boy was admitted to the Hartford Hospital for repair of a femoral nerve which had been inadvertently sectioned in the pelvis in performing an appendectomy at an outlying hospital. This had been recognized at time of surgery and one silk suture had been placed through the cut ends for purposes of later identification. The child exhibited a typical, complete femoral nerve paralysis.

Operation. On May 6, 1953, through the old appendectomy incision, the femoral nerve was found adherent to the surface underneath the internal oblique close to the inguinal ligament. A suture was found on it. It had been sectioned almost completely except for a little part of the neurolemma. The two main roots of the nerve were identified and followed upward. The two distal segments of the femoral nerve were also exposed and followed to free the nerve as much as possible. The thigh was then flexed on the abdomen at an angle of 90° to allow suture and diminish tension. The severed part was resected with a razor blade and suture of the nerve was carried out using fine tantalum wire anteriorly and posteriorly. A stay suture of tantalum was also inserted between the bifurcations of the roots and the division of the femoral nerve itself. The approximation of the nerve seemed satisfactory. The wound was closed in layers using silk. In order to preserve the suture of the nerve, a spica cast was applied with the thigh flexed on the abdomen at an angle of 90° and the leg flexed at a similar angle on the thigh. Of interest was the composition of the femoral nerve, consisting of two large trunks joining about 2 cm. above Poupart's ligament and the main trunk again dividing into two portions at Poupart's ligament.

Course. The cast was removed on June 1, 1953 and the child was allowed to be ambulatory. By June 19 he was walking unsupported. By July 20, 1953 sensation was down to 4″ below Poupart's ligament and there was some question of the slightest motion beginning to show itself in the anterior thigh muscles. By August 25, 1953, $3\frac{3}{4}$ months postoperatively, the child had good recovery indeed. There was still, however, 2 cm. of atrophy of the right thigh.

On final check up 9 months postoperatively there was only $\frac{1}{2}$ cm. difference in measurements of the right thigh in comparison with the left, with normal muscle strength and ability to walk from a squatting position.

DISCUSSION

The repair of divided femoral nerves, particularly in the pelvis, is extremely rare. The 2 patients presented above have had excellent results with, for all practical purposes, a functional recovery approaching 100 per cent. This good functional result is undoubtedly attributable in part to the relatively simple, mass functions performed by the muscles innervated by the femoral nerve and to the lack of importance in the thigh of discriminatory sensation in the areas supplied by this nerve. It is felt that repair of a severed femoral nerve in the pelvis is practical and can be carried out easily; the results are apparently excellent.

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CEREBROSPINAL FLUID RHINORRHOEA FOLLOWING REMOVAL OF AN ACOUSTIC NEUROMA

A CASE REPORT*

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An uncommon complication of the radical removal of an acoustic tumor is a cerebrospinal rhinorrhoea by way of the Eustachian tube. This sequel appears to have been recorded only by Dandy,[2] although from personal correspondence and discussion, it is apparent that many other surgeons have encountered it. Since its cure may present difficulties, the following case is reported in detail.

CASE REPORT

A. De la S. (#686149), a 29-year-old housewife, was admitted to The Johns Hopkins Hospital on Oct. 6, 1954. At the age of 12, she had had an attack of measles following which the hearing in the right ear was impaired. In the ensuing years, the auditory acuity in this ear gradually diminished. In July 1954, the patient noted formications and numbness on the right side of the face and tongue. Upon awakening on Aug. 8, 1954, she experienced severe vertigo with things whirling about in a clockwise direction; she felt nauseated and vomited. Because of persisting nausea and vomiting she remained in bed 2 weeks. About this time she noted that the right ear was deaf. A second attack of vertigo and nausea of a milder degree occurred in the latter part of August. She had no other symptoms referable to the central nervous system. Her past history was not particularly significant.

Examination. The general physical status of the patient was normal. There was slight blurring of the optic discs but because of a high degree of myopia the significance of this finding was uncertain. The extraocular movements and pupillary reactions were normal in all respects. There were hypesthesia and hypalgesia in the 2nd and 3rd divisions of the right trigeminal nerve and a slight but definite paresis of the right facial muscles. The hearing was markedly impaired and the caloric reactions were absent on the right side. Motor, sensory and reflex systems were normal. The patient's usual gait was on a broad base and when walking tandem she was unsteady and tended to sway or fall to the left.

On roentgenograms of the skull a large defect was seen in the right petrous apex (Fig. 1). On the basis of the history, findings and complete absence of acoustic and vestibular function on the right side and the marked erosion of the petrous portion of the temporal bone, a diagnosis of right acoustic neuroma was made.

1st Operation. On Oct. 10, 1954 through a vertical right occipital incision the right cerebellar hemisphere was exposed, and its lateral third was ablated to expose a firm encapsulated tumor in the cerebellopontine angle. Just anterior to the 9th, 10th and 11th nerves the tumor had eroded the petrous portion of the temporal bone over an area approximately 2 cm. in diameter. The capsule of the tumor was cut from the margin of the internal acoustic meatus, gradually dissected from the adjacent cerebellum and with little difficulty was removed in one piece. Tumor tissue in the internal acoustic meatus was curetted out and the cavity, which measured almost 2 cm. in diameter, was thoroughly cauterized with the electrosurgical

* Presented at the annual meeting of the American Academy of Neurological Surgery, October 28, 1955 at Hot Springs, Virginia.

unit and bone wax was applied to its roughened surface. The wound was closed without drainage.

Postoperative Course. The patient's convalescence was uneventful until, on October 18, watery discharge from the right nostril was noted. During the next few days varying amounts of fluid drained from the nostril, on some occasions there being no drainage and again considerable. On October 25 the fluid collected had 68 mg. per cent of reducing substance.

2nd Operation. Because of the persistent drainage the wound was explored on Oct. 26,

FIG. 1. Photograph of a roentgenogram of the skull to show the extensive erosion of the right petrous pyramid. The pneumatization of the mastoid is well shown.

1954. Under Pentothal anesthesia a vertical incision was made in the right temporal region and carried down to the squamous portion of the temporal bone. The bone was perforated and the opening was enlarged. Upon elevating the dura mater from the petrosal ridge a darkened area of thin bone—the tegmen tympani—was seen overlying the dilated internal acoustic meatus. When this was ruptured a large cavity in the petrous tip was exposed in which could be seen the lateral aspect of the pons and cerebellum. In the base and walls of this cavity were numerous large mastoid air cells which were plugged with bone wax and Gelfoam.

Course. The patient recovered from this operation quite well and had no rhinorrhoea. She was discharged from hospital on Nov. 4, 1954, but within a week was readmitted for a lateral tarsorrhaphy because of a corneal ulceration. Two days later fluid was seen draining

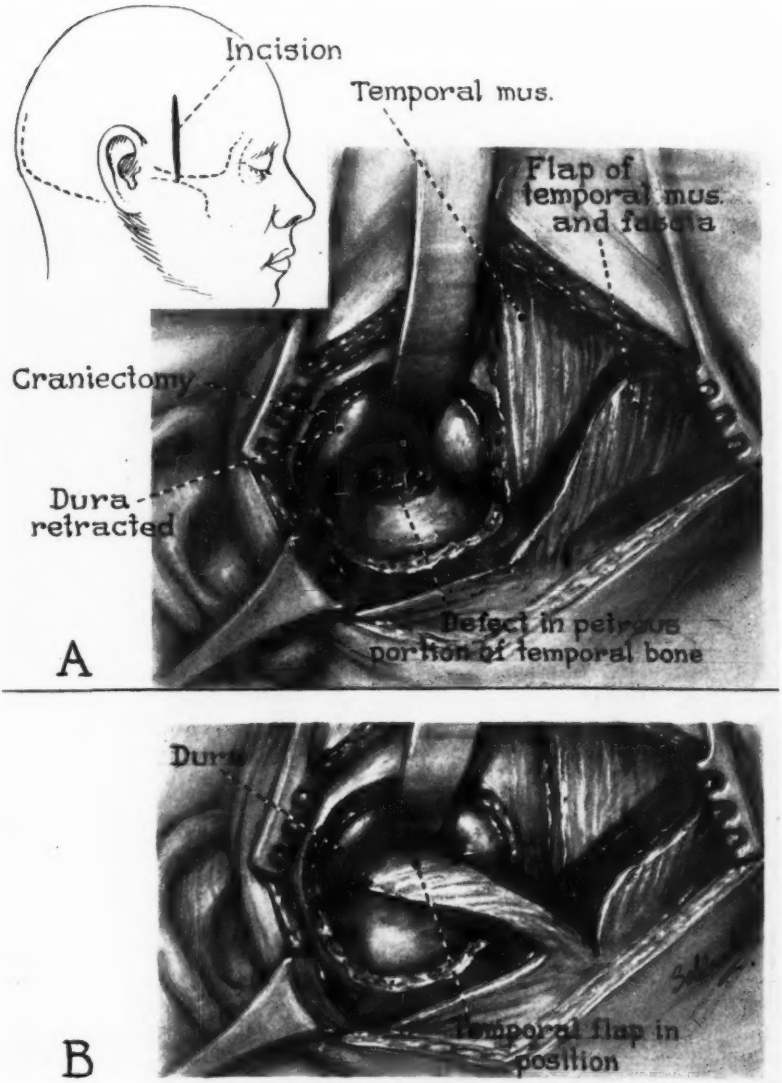

FIG. 2. (A) Sketch to indicate the operative incision and approach to the temporal tip through the middle fossa. (B) The muscle flap has been cut from the temporal muscle and aponeurosis and swung across the floor of the middle fossa to the defect in the temporal tip.

from the right nostril. After consultation, Dr. Walter Loch, of the Ear, Nose and Throat Service, decided to explore the mastoid with the intent of plugging the mastoid air cells leading to the middle ear and in this way eliminate the leak. This was done on Nov. 16, 1954. For 2 or 3 days there was no drainage but on November 21 the rhinorrhoea was noted again. The mastoid wound became infected and cultures grew out a hemolytic Staphylococcus aureus insensitive to penicillin, Terramycin or Achromycin and only slightly sensitive to Aureomycin and moderately sensitive to erythromycin. On December 4 the patient was complaining of severe headache. Her temperature had risen to 101.4°F. and her neck was

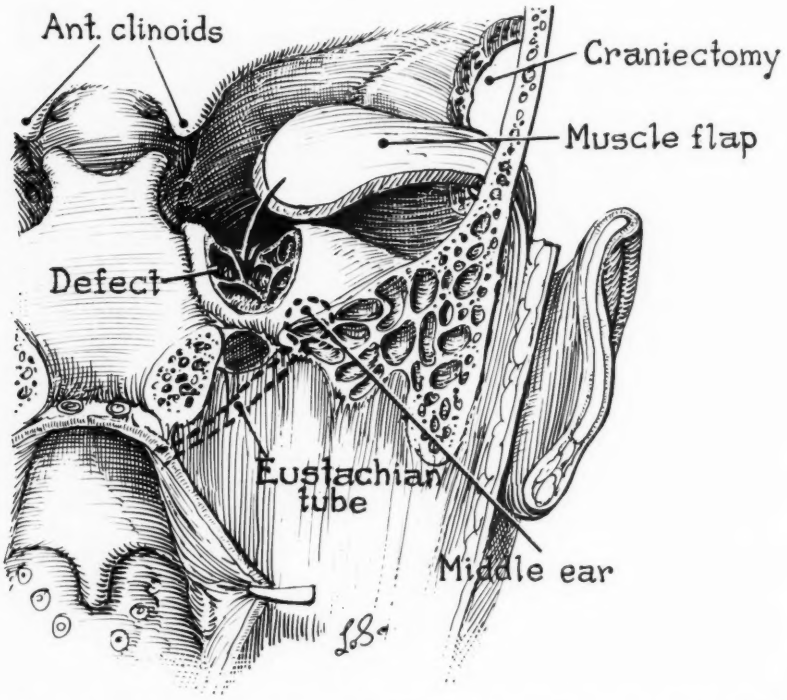

FIG. 3. Vertical section looking at the posterior aspect of the base of the skull to show the erosion of the internal auditory meatus, the extensive pneumatization of the petrous tip and the method of plugging the osseous defect with the muscle flap.

stiff. A diagnosis of meningitis seemed obvious and the mastoid wound was reopened. It was found that the purulent drainage came from a soft-tissue abscess. The mastoid cavity previously filled with Gelfoam was cleaned out but there was no purulent discharge in that area. The wound was packed with iodoform wax which stopped the cerebrospinal fluid discharge from the pyramidal region of the mastoid.

The patient recovered rapidly from this procedure but continued to have a low-grade fever. Upon lumbar puncture on December 5 the fluid was found to contain 247 lymphocytes and 13 polymorphonuclear leukocytes.

Because the drainage was continuing both from the mastoid and nose, and since there was frank infection in the mastoid it was imperative that a radical attempt be made to seal off the intracranial cavity. There seemed little to be gained from attempting to plug the mastoid itself.

3rd Operation. Under local anesthesia on Dec. 12, 1954 the previous defect in the temporal bone was uncovered and the dura mater was elevated from the floor of the middle fossa. This gave a good exposure of the defect in the dura mater and petrous tip (Fig. 2). A tennis racket-shaped piece of temporal muscle, approximately 2 cm. in diameter, attached by the aponeurosis and fascia was dissected out and swung across the floor of the middle fossa and down into the dehiscence in the petrous tip (Fig. 3). This muscle was packed in the defect and seemed to cover completely the mastoid air cells. The aponeurosis was then sutured to the margin of the dural defect in order to prevent it from pulling out with contraction of the temporal muscle. The temporal lobe was allowed to fall back into place and the muscles were sutured in anatomic layers.

Course. The patient's condition improved quite rapidly. She was given Crysticillin in addition to erythromycin and her temperature gradually decreased. There was a moderate but decreasing amount of watery drainage from the mastoid wound for approximately 3 days. On December 23 the patient was quite well and was discharged from the hospital. She had no further rhinorrhoea but returned on Feb. 1, 1955 for a hypoglossofacial anastomosis and a plastic operation upon the right side of the face.

The patient was last seen on Feb. 27, 1955 at which time she had no complaints. The right side of her face felt a bit numb, a feeling that had been present since her plastic operation. She had very slight difficulty in swallowing and since her last operation at times had to use milk or water to wash down her food. All of her wounds had healed. She had considerable drooping of the right side of the face and the eyelids did not quite approximate when an attempt was made to close them. The masseters, however, contracted well. The corneal sensation was good. The right ear was completely deaf. Otherwise the neurological findings including the gait were normal. The patient has written several times since her discharge stating that she has been quite well and has had no further complaints.

DISCUSSION

There seems little doubt that the cerebrospinal fluid leak in this case resulted from the extensive erosion of the tumor into and the subsequent operative opening of the mastoid air cells. That this complication occurs so rarely is probably because of the fact that pneumatization of the mastoid tip is not common,[1] and that extensive erosion of the internal acoustic meatus occurs in only a small percentage of cases of acoustic tumors. In the usual extirpation of an acoustic tumor, air cells in the petrous tip are not entered; only in the exceptional case is there such pneumatization in this area that erosion of the petrous tip allows the cerebrospinal fluid of the pre-pontine cistern free access to the tympanitic antrum, middle ear and Eustachian tube. Unless there is a defect in the tympanic membrane the fluid will drip into the nasopharynx, and, if the head is flexed, into the ipsilateral nares. This paradoxical rhinorrhoea has been commented upon by previous writers on the subject of rhinorrhoea and otorrhoea.[2] The fact that a fluid level is usually present in the middle ear as it was in the present case should assist in the differential diagnosis.

The treatment of this paradoxical rhinorrhoea secondary to removal of an acoustic tumor is not simple. The prophylactic sealing of the mastoid air cells with bone wax probably prevents many such leaks, but in some cases air sinuses make watertight waxing impossible. Because of the inaccessibility it is difficult, if not impossible, to seal off the region of the internal acoustic meatus with a piece of fascia such as is usually advocated for a rhinorrhoea through the cribriform plate. Nor does Gelfoam give an adequate seal.*

* Dr. Joseph P. Evans in a discussion of this paper stated that he had plugged a leak in the petrous pyramid with a muscle pedicle flap from the posterior muscles of the neck and placed a piece of fibrin film over it for a dural substitution. Dr. George Baker referred to a case in which the hole was plugged successfully with "Ivalon sponge."

There remain at least two other routes of attack. Through a mastoid approach the tympanic cavity might be opened and mastoid cells or the orifices of the Eustachian tube blocked with bone wax, Gelfoam, iodoform gauze or sutures. This technique is not entirely suitable since fluid has access to the middle ear and mastoid region. It was not successful in Dandy's case nor in this case. The other approach is through the tegmen tympani. By this route it is possible to pack the entire tympanic cavity and defect in the petrous tip with muscle. In the usual case a free muscle graft might be an adequate pack, but in the presence of infection a viable graft is considered more desirable. A few sutures in the aponeurosis and dura mater over the petrous tip will hold the transplant for a few days until fibrous tissue has fixed it in place.

SUMMARY

Following a radical excision of a right acoustic neuroma, a rhinorrhoea developed. In spite of two attempts to close the fistula from the petrous tip to the Eustachian tube by bone waxing and plugging the mastoid cells with Gelfoam the cerebrospinal fluid leak continued. Infection developed in the mastoid wound, which had been made in an attempt to seal off the air cells, and meningitis ensued. Through a temporal approach a flap of muscle was inserted into the tympanic antrum and defect in the petrous tip and sutured to the dura mater. The leak ceased and the patient made an uneventful recovery from the meningitis.

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PERSISTENT CEREBROSPINAL RHINORRHEA ORIGINATING IN A FRACTURE THROUGH THE PETROUS BONE AND CURED BY MUSCLE GRAFT

REPORT OF A CASE

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In the majority of cases, persistent cerebrospinal rhinorrhea results from defects in the floor of the anterior fossa. However, in a few instances it occurs through the petrous pyramid or mastoid bone, the fluid draining into the middle ear and through the Eustachian tube into the nasopharynx.[4,6–8] Defects in the temporal bone may be produced by trauma, surgical or accidental, or by erosion of tumors.

Persistent cranionasal or cranio-otic fistulae require closure in order to prevent inevitable intracranial infection and severe headache or pneumatocele.[4–7,15,16] In 1926 Dandy[7] clearly pointed out that suture or patching of the dura mater was essential for cure. Various methods of accomplishing this have been tried, the success that has been attained having been proportional to the adequacy of reestablishment of dural continuity.[1–3,5,6,9–14]

The following case is considered sufficiently unusual to warrant recording.

CASE SUMMARY

J.H., a 37-year-old man, was admitted to the Albany Hospital on Aug. 20, 1954. Two days previously he had fallen a distance of 20 feet, striking his head. He had been unconscious for 45 minutes, and was dazed for 4 hours. He vomited several times and had bled from the nose. Because of persistent drowsiness he was brought to the hospital. He was somewhat confused and complained solely of numbness of the right side of the face.

Examination. He was a well developed, well nourished man who was awake but confused. Temperature was 98.5°, respiratory rate 18, pulse rate 52/min. and blood pressure 110/70. There was a sutured laceration in the right frontal area. No bony deformity of the head or neck was found but the neck was rigid. The right tympanic membrane was blue and bulging. There was no active bleeding from the nose or throat. Except for bradycardia the chest and cardiovascular systems were normal. The abdomen was negative.

Fig. 1. Roentgenogram of base of skull showing wide fracture (retouched) through right petrous pyramid.

* Deceased February 15, 1956.

His sense of smell was intact. The pupils were equal and reacted. Abnormal neurological findings were: hypaesthesia on the right side of the face, weakness of all muscles of expression on the right, and deafness of the right ear.

Roentgenograms of the skull disclosed a fracture line extending from the right parietal bone through the floor of the middle fossa and across the petrous pyramid to the posterior fossa (Fig. 1).

Course. During the next 4 days he was maintained on antibiotic therapy and observed closely. His confusion disappeared but the bradycardia persisted. On August 24 he sat up for the first time and pink fluid drained out of the right nostril. This was followed by severe

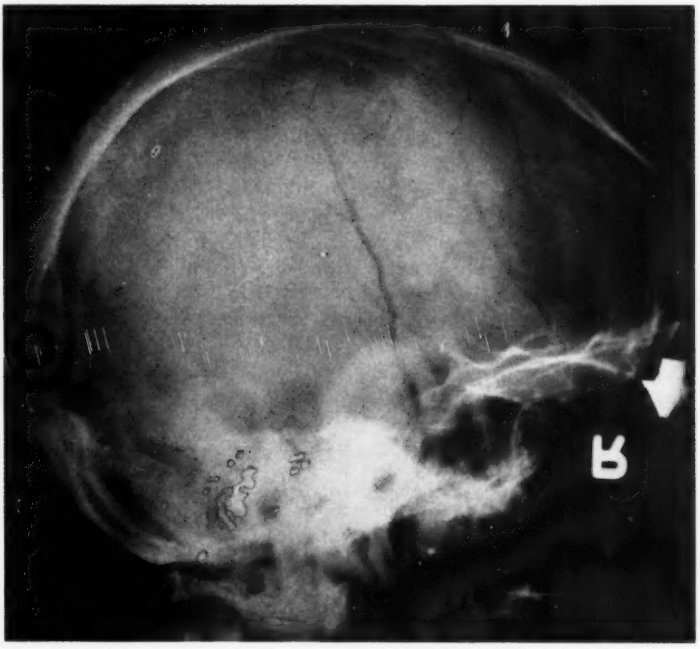

FIG. 2. Lateral roentgenogram of skull (retouched) taken following instillation of pantopaque into cisterna magna. Radio-opaque material is visible in the right posterior fossa and right side of the nasopharynx.

headache. Analysis of this fluid revealed the presence of 45 mg. per cent glucose. Nasopharyngoscopy showed fluid draining out of the ostium of the right Eustachian tube. The temperature remained normal.

On September 3, because of persistence of the drainage and in an attempt to discover whether the leak was anterior or posterior to the petrous ridge ½ cc. of pantopaque was placed in the cisterna magna and the patient was kept on the right side. Roentgenograms (Fig. 2) revealed radio-opaque material in the posterior fossa and nasopharynx but none in the middle fossa.

Operation. On Sept. 7, 1954, because of failure of the fistula to heal spontaneously, a right suboccipital craniectomy was performed and the posterior surface of the petrous pyramid was visualized. At the junction of the middle and inner thirds a wide tear in the dura mater could be seen. This was just in the region of the cisterna lateralis, extending from the internal auditory meatus vertically to the petrous ridge. A piece of muscle was placed in and

over the defect. Using the electrocoagulation current the muscle was burnt and appeared to stick to the surrounding dura mater.

Postoperative Course. For 4 days a very small amount of cerebrospinal fluid drained from the nose. When discharged on Sept. 21, 1954, he was ambulatory. He was deaf in the right ear. Taste sensation over the entire tongue was normal. There was still some hypaesthesia over the lower part of the right side of the face. When seen 5 months later the facial weakness had almost entirely disappeared. Hearing in the right ear had not returned. All cerebrospinal fluid drainage had ceased. Headaches had disappeared and he had returned to work.

SUMMARY

A case of persistent cerebrospinal rhinorrhea caused by cranio-otic fistula is presented. The problems posed concerned the radiographic demonstration of the fistula's origin in the posterior rather than the middle fossa. The difficulty of suturing a dural patch in this region was obviated by the use of muscle, tightly wedged in and rendered more adherent by the electrocautery.

This method would no doubt be applicable to persistent fistulae in this area, such as those caused by extensive curettement of the internal auditory meatus for the removal of tumors.

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ABSCESS FORMATION IN THE PITUITARY FOSSA ASSOCIATED WITH A PITUITARY ADENOMA

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A pituitary adenoma with abscess formation is an extremely rare finding, only two cases having been found recorded in the literature. Whalley,[2] in 1952, reported the case of a patient who presented a picture of fulminating meningitis, and who died before a diagnosis could be made. At autopsy an abscess in a pituitary adenoma was found as the cause of the acute meningitis.

Asenjo[1] described a case in which the presence of an hypophysial adenoma with typical clinical and radiological findings was detected after the patient had been treated for acute sinusitis, and complained of sudden visual disturbance. Operative treatment was successful.

In the case reported here, as in Whalley's[2] case, the presenting symptoms were those of acute intracranial pathology. The pituitary adenoma which was present was diagnosed only during the course of investigations which were performed in order to ascertain the cause of his acute illness.

CASE REPORT

H.P., a white male aged 37 years, consulted his doctor on June 4, 1953, complaining of a very severe left-sided headache, which came on at a moment's notice as he was closing the door of his car. As the excruciating headache persisted, he was referred to a neurologist 2 days later. At this time, no abnormalities in the general or neurological examinations were evident, and because of the patient's almost "wild" behaviour, caused by the severe headache, he was admitted to hospital for observation, with doubt as to organic aetiology, and with suspicion of a psychosis.

After admission photophobia developed and, 2 days later, there was a partial 3rd nerve palsy on the left side, which progressed rapidly to complete paralysis within 24 hours. The development of these signs left no doubt as to the presence of an organic lesion, and in spite of the absence of rigidity of the neck, an aneurysm was suspected. Lumbar puncture yielded clear, colourless spinal fluid under normal pressure. There was no rise in the protein content, and 5 lymphocytes were present.

The patient was transferred to the Neurosurgical Unit.

Examination. He complained of persistent excruciating headache and marked photophobia. Blood pressure and pulse rate were normal. Temperature was slightly raised. The optic discs were normal. To rough confrontation there was no impairment of the visual fields. These could not be accurately tested because of the rather poor co-operation of the patient, who was in a distressed condition. There was no evidence of other neurological abnormalities.

Plain roentgenograms of the skull revealed a grossly enlarged pituitary fossa, with thinning of the posterior clinoid processes. There was complete erosion of the posterior portion of the sella turcica. A calcified pineal gland was observed in the postero-anterior view, and was centrally situated. Roentgenograms of the sinuses showed no definite abnormality. Minimal changes were present in the right antrum which were considered of doubtful pathological significance.

In spite of the erosion of the pituitary fossa, suggesting the presence of a pituitary ade-

noma, an aneurysm had to be excluded in view of the history of sudden onset of headache, and the 3rd nerve paralysis. Left percutaneous carotid angiography was performed under general anaesthesia. There was no evidence of an aneurysm. Following angiography, an air-encephalogram was performed which revealed a normal ventricular outline. A diagnosis of pituitary adenoma was made.

Operation. On June 16, 1953, under general anaesthesia, the region of the optic chiasm was exposed via a left transfrontal approach. The bluish capsule of a tumour mass was evident and, after coagulation of the capsule, an incision was made and the tumour was removed by suction and spoon. After the major portion of the tumour had been removed, the bed was scraped with a spoon, and a sudden welling of thick yellow pus in the tumour bed occurred. The total volume of pus aspirated was 5 cc. Complete evacuation of pus and remaining neoplastic tissue was performed, haemostasis was effected, and patties soaked in 200,000 units penicillin were packed into the fossa for several minutes. Closure was then performed in the usual manner.

Histologically the tumour was a chromophobe adenoma.

Culture of the pus yielded a growth of Staphylococcus aureus which was sensitive to the five antibiotics tested.

Course. The patient received intramuscular penicillin and streptomycin, and daily intrathecal penicillin injections of 25,000 units for 3 days. In the first 3 days, the temperature fluctuated between 100° and 100.3°F., then settled to normal. Apart from this, the patient made an uneventful recovery. Relief from the severe headache was immediate. The 3rd nerve palsy started improving after 24 hours, and progressed to almost complete recovery after 10 days. At this time there was slight residual weakness causing occasional diplopia.

The patient was discharged on the 23rd postoperative day, symptom free. Vision on the right side was 6/9, and on the left 6/12. The visual fields were full. The 3rd nerve palsy had cleared up completely, no diplopia being complained of.

The patient was kept under observation and reported every 3 months. The visual fields remained normal, and visual acuity improved. The only complaint was a feeling of tiredness.

On Sept. 21, 1953 he suffered a severe headache, which alarmed both the patient and his wife, in view of the similarity to the beginning of his initial illness. Examination revealed normal neurological findings, but a slightly raised temperature. Roentgenograms of the skull and sinuses demonstrated changes characteristic of a pansinusitis. This condition rapidly improved and cleared up on antibiotic therapy.

Although the roentgenograms prior to operation showed no definite pathology of the sinuses, apart from minimal changes in the right antrum, the subsequent pansinusitis strengthened the presumption that the suppuration in the pituitary fossa, which was found at operation, was caused by extension from a sphenoidal sinus infection.

Because of the persistent complaint of tiredness, a further assessment of endocrine function was made and, although no evidence of gross pituitary deficiency was revealed, he was given a small maintenance dose of cortisone and testosterone. After this the tiredness disappeared and further, the patient volunteered the information that his libido had improved, although this loss had not been mentioned prior to the investigations. Periodic follow-up examinations have revealed no abnormalities and the patient is symptom free.

DISCUSSION

Cases of inflammatory processes and abscess formation in the pituitary gland have been described in the literature, and have been discussed in an article by Asenjo.[1] His case, that of Whalley and the case described here are, however, the only ones known in which abscess formation and pituitary adenoma coexisted.

In the present case the abscess in the pituitary fossa, which undoubtedly originated from an infection of the sphenoidal sinus, caused the acute presenting symptoms which led to the diagnosis of a pituitary adenoma. The fairly rapid onset of the 3rd nerve paralysis must have been caused by excessive pressure in a lateral direction, extending to the cavernous sinus with compression of the 3rd nerve.

As mentioned, this case bears a similarity to that described by Whalley,[2] in that the onset was sudden, and not actually associated with symptoms of a pituitary tumour. In the case of Whalley, however, the clinical picture was that of a fulminating meningitis, which was originally thought to be secondary to a cavernous sinus thrombosis. Bilateral blindness developed with great rapidity, and the patient died within 12 hours of admission to hospital. Autopsy showed a tumour occupying the sella turcica, with an abscess occupying one whole side of the mass. No evidence of a break-through into the subarachnoid space was found at autopsy, which makes the mechanism of spread of the meningitis rather difficult to explain.

Asenjo's[1] patient had been treated for a frontomaxillary sinusitis which had cleared up but, because of the persistence of the complaint of visual disturbance which had commenced with the sinusitis, he was referred to the Institute of Neurosurgery, and a diagnosis of pituitary adenoma was made from the patient's clinical symptomatology, radiological findings and the visual field charts which showed a typical, complete bitemporal hemianopia. At this time there were no further signs of an acute inflammatory condition present.

SUMMARY

A case is presented of pituitary adenoma with abscess formation in the pituitary fossa.

The clinical symptomatology was unusual, as the patient did not present the classical symptoms or signs of an hypophysial tumour, and diagnosis of the adenoma was made on the radiological findings.

The presenting feature was the acute onset of symptoms of meningeal irritation which, combined with the subsequent onset of a 3rd nerve paralysis, were at first suggestive of an aneurysm with subarachnoid haemorrhage.

Only two other similar cases have been found in the literature. Whalley's[2] patient presented symptoms of a fulminating meningitis, and the diagnosis was made at autopsy. In Asenjo's[1] case, the associated abscess was a complicating factor in a symptomatology that was diagnostic of a pituitary adenoma.

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ARTERIOVENOUS ANEURYSM OF THE POSTERIOR FOSSA IN AN INFANT

REPORT OF A CASE

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Vascular malformations of the posterior fossa in infants are extremely rare. Dandy[3] reported an infant of 1 month with an angioma of the posterior fossa with extracranial and intracranial extension and development of hydrocephalus who subsequently died on attempted removal of the tumor. Richardson and Bagnall[8] described a case of angioma of the posterior fossa in a male 23 years of age whose symptoms were first noted at the age of 7 years. The patient died at the age of 23 years, and at autopsy an angiomatous malformation of the posterior fossa was found. Grotts[4] reported a 2-year-old child, who died from a spontaneous hemorrhage, with a cavernous hemangioma of the pons found at autopsy. Olivecrona and Riives[6] reported 60 cases of arteriovenous aneurysms out of 3206 verified tumors of the brain. Of these 60 aneurysms, only 2 were in the posterior fossa, and these 2 were in males aged 39 and 50 years. Clement *et al.*[1] reported 2 cases of arteriovenous aneurysms in infants aged 5 weeks and 8 months. The first case was that of an infant who died at the age of 5 weeks; autopsy revealed an ovoid aneurysmal dilatation at the junction of the free edge of the falx cerebri with the tentorium cerebelli. The second child had dilatation of the right sinus rectus, torcular Herophili, and lateral sinus as well as an arteriovenous aneurysm of the ampulla of Galen. These were proved by angiography. The findings were verified at autopsy.

The following case report is of a 2-month-old white male infant who had a spontaneous subarachnoid hemorrhage from an arteriovenous aneurysm of the posterior fossa which was diagnosed by vertebral angiography and successfully removed.

CASE REPORT

JG #174594. A 2-month-old white male infant was admitted to the Baptist Memorial Hospital on Nov. 18, 1954. He was referred to this hospital by Dr. J. N. Robinson, Columbus, Miss., and Dr. J. R. Mullens, Jr., West Point, Miss. The infant was a full-term fetus delivered under general anesthesia with no complications. He was apparently normal until 3 weeks before admission when he was said to have had a convulsion with loss of consciousness. He was taken to Dr. Mullens, who referred the child to Dr. Robinson. A spinal puncture yielded xanthochromic fluid. The examination prior to this time had shown that the anterior fontanelle was tense and bulging. A pneumoencephalogram was performed by Dr. Robinson which showed slight ventricular enlargement and absence of air over the cerebral hemispheres.

Examination. The head was obviously enlarged. The occipitofrontal circumference of the skull measured 41 cm.; the anterior fontanelle was large and bulging. Lumbar puncture revealed slightly xanthochromic spinal fluid with 164 mg. per cent of total protein. Ventricular fluid was colorless.

Ventriculography revealed the lateral and 3rd ventricles to be symmetrically enlarged

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with no filling of the 4th ventricle or the aqueduct of Sylvius. The enlargement of the ventricles was noted to be greater than on the previous encephalogram. Two days following this pneumoencephalography was performed with 50 cc. of air to demonstrate the patency of the 4th ventricle and aqueduct. The lateral ventricles were well filled, as was the 3rd ventricle. Views of the 4th ventricle and aqueduct were not completely satisfactory. There was very little air over the surface of the cerebral cortex.

Course. The child was discharged Nov. 26, 1954 with a diagnosis of hydrocephalus, most probably caused by failure of the subarachnoid channels to develop properly. The parents were instructed that pneumoencephalography was to be repeated in 1 month.

Readmission. The child was readmitted on Jan. 3, 1955 because of rapid enlargement of the head from 42 cm. to 47 cm. No other significant abnormality had been detected. Pneumoencephalography was repeated. This study revealed an obstruction in the 4th ventricle and

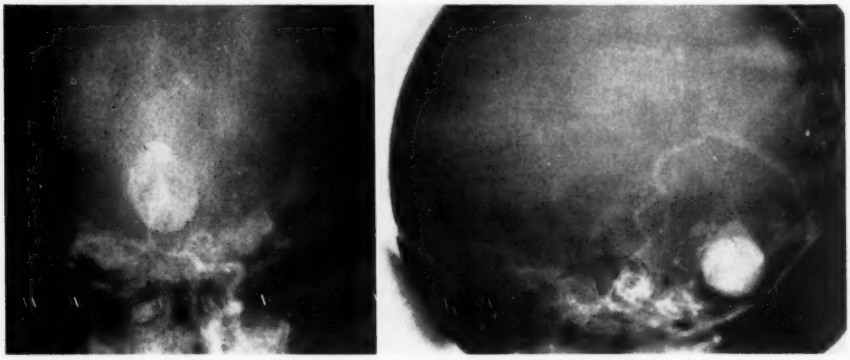

FIG. 1. Vertebral angiogram. (*Left*) Anteroposterior view showing the aneurysm filling from the left vertebral artery. (*Right*) Lateral view showing the aneurysm in the posterior fossa.

poor filling of the lateral and 3rd ventricles. Air in the 4th ventricle seemed to outline a mass.

1st Operation. On Jan. 11, 1955 a suboccipital craniectomy was performed through a midline incision. Upon opening the dura mater, a smooth, white, glistening mass presented itself in the midline, approximately 3 cm. in diameter. A 25 gauge needle was inserted into the mass and arterial blood was obtained. Palpation of the mass then revealed an obvious pulsation and it was immediately apparent that a vascular malformation was present. Because of the limited exposure plus the fact that the origin of the vascular malformation was unknown, no definitive procedure was done and the operation was terminated.

Course. On Jan. 17, 1955 left vertebral angiography was performed with 35 per cent Diodrast. A large, midline aneurysmal sac was noted in the posterior fossa as had been seen at surgery (Fig. 1). It was thought that the feeding vessel was the left vertebral artery with drainage into the straight sinus.

2nd Operation. On Jan. 25, 1955 a suboccipital craniectomy was performed through a horseshoe-shaped incision. The aneurysmal mass was satisfactorily exposed. It was noted that the left vertebral artery divided before entering the large aneurysmal sac. Each division was clipped with an Olivecrona type of clip. No ill effects were noted and the aneurysm ceased to pulsate; however, this was only temporary and the aneurysm again started to pulsate in a few moments. The entire left cerebellar hemisphere was cystic and degenerative in nature, and the major portion of it was occupied by a huge angiomatous mass. The angiomatous mass together with the large aneurysm was dissected free and carefully divided from its vascular attachments. It was thus removed en bloc. After the removal, very little tissue remained of the left cerebellar hemisphere.

Course. The infant tolerated the procedure quite well. When discharged from the hospital his fontanelle had remained soft as well as the site of operation.

Pathologic Report. Gross. The fixed specimen is an irregularly shaped mass of tissue measuring approximately 4 cm. by 3.8 cm. by 3 cm. (Fig. 2). The main portion of this mass is made up of shaggy, dark-red tissue which has a soft, spongy consistency and is traversed by numerous small vascular spaces. Situated on one side of this mass of hemorrhagic tissue is a cystic structure which is round and measures 2.5 cm. in diameter. Several small vessels are seen entering this cyst, which has a smooth lining and a thin wall of rather firm, white tissue. It is filled with a clot of dark, red blood. There are five small openings into this cystic space, communicating with the hemorrhagic tissue outside. These openings are from 1 to 2 mm. in diameter.

Microscopic. Sections of the solid portion of the lesion reveal that it is composed predominantly of masses of small capillaries. Often these form ill-defined regions of small con-

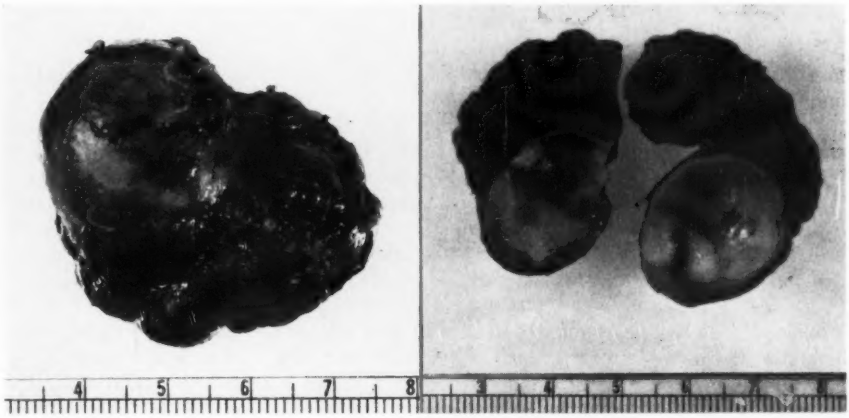

FIG. 2. Photographs of pathological specimen (fixed in formalin). (*Left*) Entire angiomatous malformation showing the large aneurysm in relation to the left cerebellar tumor mass. (*Right*) Specimen divided transversely to show the aneurysm and its related vessels.

gested capillaries and are associated with some hemorrhage. Elsewhere they occur as rounded to ovoid, discrete masses containing similar capillaries. Most of the capillaries are quite well-formed and have small to slightly dilated lumens. They are lined by flat to slightly plump endothelium in which no mitotic figures were found after careful search. Only rarely small foci of endothelial cells with formed lumens are present. Portions of edematous cerebellar tissue are found frequently within the main mass of tumor. This cerebellar tissue is readily recognized, especially by portions of the molecular layer persisting. As a whole, there are relatively few macrophages present, but there is a moderate amount of hemorrhage.

Sections taken through the wall of the grossly described cyst reveal that it is made up of thickened muscular tissue which is somewhat edematous. It is lined by endothelium. The wall appears to be of uniform thickness and special stains fail to reveal any elastic tissue in it. These special stains confirm the presence of scattered, small, usually well-formed arteries, but these do not appear to be increased in number or abnormal in appearance. In a few places the capillary hemangiomatous elements are seen adjacent to the wall of the main cyst.

Because the main cystic mass lacks any elastic tissue in its wall, it is believed that this is derived from venous tissue. It is not clear just how this has been formed, but it is suspected that it is the result of the tumor creating an arteriovenous aneurysm, perhaps by destruction of the intervening tumor tissue.*

* Pathologic description by Dr. M. L. Trumbull, Chief, Department of Pathology, Baptist Memorial Hospital, Memphis, Tennessee.

Subsequent Course. The patient has been seen subsequently on a return visit to the clinic. The anterior fontanelle has remained soft and the head has not enlarged. The child has remained asymptomatic.

DISCUSSION

A careful search of the literature, although admittedly incomplete, has failed to reveal any case similar to this one in which an arteriovenous aneurysm of the posterior fossa has been successfully diagnosed and removed in an infant. The diagnosis in this case could have been made at the age of 5 weeks when the child apparently had his first spontaneous subarachnoid hemorrhage. This was evidenced by xanthochromia of the spinal fluid following his first seizure. Patterson[7] in discussing the diagnosis of posterior fossa angiomas has emphasized that the symptomatology is almost identical to that produced by supratentorial angiomas. Cerebellar symptoms together with obstructive hydrocephalus suggest a tumor in the posterior fossa in most instances. The presence of subarachnoid hemorrhage suggests a vascular anomaly of the posterior fossa.

The pathologic report of the specimen describes the large aneurysmal sac, the wall of which is composed of muscular tissue. The fact that special stains failed to reveal any elastic tissue in the wall is significant and probably means that the sac is of venous origin. Surrounding the larger aneurysmal sac were masses of small capillaries, some of which were arterial. The stroma of the tumor mass was composed of edematous, cerebellar tissue. The microscopic picture of this specimen corresponds closely with that of a 16-year-old boy described by Olivecrona and Lysholm[5] (Case 65). The tumor was exposed by a suboccipital approach. It appeared to be inoperable, the boy subsequently died from hemorrhage and at autopsy an angioma arteriale was proved to be present. As in our case, the vessels were separated by cerebellar tissue; however, the main vessel in their case was arterial as shown by the presence of elastic tissue in the wall with a well-formed elastica interna. The presence of cerebellar tissue between the vessels, as suggested by Cushing and Bailey,[2] distinguishes the lesion as an arteriovenous aneurysm, as opposed to a true neoplasm, such as a hemangio-endothelioma of the cerebellum.

SUMMARY

A case report of a 2-month-old infant with an arteriovenous aneurysm of the posterior fossa has been described. The lesion was correctly diagnosed and treated surgically. No other case such as this has been noted in the literature.

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MENINGIOMA OF THE INTERNAL AUDITORY MEATUS IN A CHILD 3 YEARS OLD*

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(Received for publication January 3, 1956)

Intracranial meningiomas are very rare in childhood. This fact is well expressed in series like that of Bailey *et al.*[1] who, among 100 intracranial tumors in children, found only 2 typical meningiomas and one of them in apparent association with von Recklinghausen's disease. Keith *et al.*[8] reported only 3 among 606 children below the age of 15 years. Cuneo and Rand,[4] in a series of 83 intracranial tumors in childhood, had 2 meningiomas, 1 of them in a 3-month-old baby with a calcified lesion which suggested that the tumor may have existed prior to birth. More recently, Ingraham and Matson[7] reviewed 313 intracranial tumors in children with only 1 meningioma among them. This was a parasagittal frontal tumor in a 7-year-old boy.

In the series of meningiomas of the posterior fossa reported by Cushing and Eisenhardt,[5] Campbell and Whitfield,[2] Petit Dutaillis and Daum,[10] D'Errico,[6] Castellano and Ruggiero,[3] Russell and Bucy,[11] and Markham *et al.*[9] the youngest patient was a 9-year-old boy[10] and the tumor was attached to the vicinity of the condyloid foramen. In the literature reviewed, the youngest patient with a meningioma of the internal auditory meatus was 15 years old.[9] Therefore, it seems interesting to report this case of a 3-year-old girl with a meningioma in this region.

CASE REPORT

G.A.C., born Sept. 24, 1951, was brought to us for the first time on May 26, 1955, because of marked weakness of the muscles of the left side of the face. This weakness was first noticed by the mother, who is a dentist, in January, 1955, but it probably had existed before that time because, in looking over previous photographs, we observed in a picture taken in February, 1954 some facial asymmetry with facial folds less marked on the left. Undoubtedly, this weakness had slowly increased since January, 1955, and, thus, at the beginning of May, the girl could not close her left eye.

A small angiomatous nevus in the midline of the occipital region and another one in the right hemithorax present since birth were destroyed with carbonic ice when the girl was 3 or 4 months old. Otherwise, the development of this child had been normal.

Examination. There was an almost complete left facial paralysis of peripheral type and, on rough testing, the impression was gained that the girl could not hear with her left ear.

Otological examination performed by Dr. Jorge de Cárdenas disclosed deafness of left ear and no response to caloric stimulation on the left with normal response on the right.

Plain radiograms of the skull on May 31, 1955 showed in the Towne position a definite enlargement of the left internal auditory meatus with thickness of its posterior margin (Fig. 1). This enlargement was confirmed by tomographic views in the usual anteroposterior position.

A diagnosis of left cerebellopontine angle tumor was made and operation was decided upon without the aid of lumbar puncture or air studies.

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* Presented at the annual meeting of the Neurosurgical Society of America, Miami, Florida, January 18–21, 1956.

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Operation. On June 20, 1955, at the Clínica Miramar, under general anesthesia with intravenous rectal Pentothal and endotracheal nitrous oxide, a left hemisuboccipital craniotomy was performed through a straight lateral incision as advocated by Bucy. The patient was placed in a lateral, semivertical position with the left side up. A round, hemispherical tumor, about 2 cm. in diameter, with a slightly pink, granular surface was encountered in the region of the internal auditory meatus. The 7th and 8th cranial nerves penetrated the tumor mass

FIG. 1. (*Above*) Plain radiogram in the Towne position, showing enlargement of the left internal auditory meatus and thickness of its margin. (*Below*) Postoperative film.

through its middle portion with no apparent plane of cleavage. It was soon realized that the nerves had to be sacrificed and therefore they were sectioned in order to allow complete gross removal of the tumor which was accomplished with the aid of pituitary rongeur and curette. The internal auditory meatus was found markedly enlarged and the tumor appeared attached to the dura mater of its posterior edge. The dura mater in all this region was resected and, although there was no macroscopic evidence of invasion of the bone, the edges of the internal auditory meatus were removed with the aid of rongeurs and its bottom was curetted and cauterized. The area of bone removal can be seen in the plain radiograms taken postoperatively (Fig. 1). There was no need to resect any portion of the cerebellar hemisphere and closure was carried out with silk sutures in the usual manner.

Postoperative course was uneventful. The facial paralysis that was almost complete before

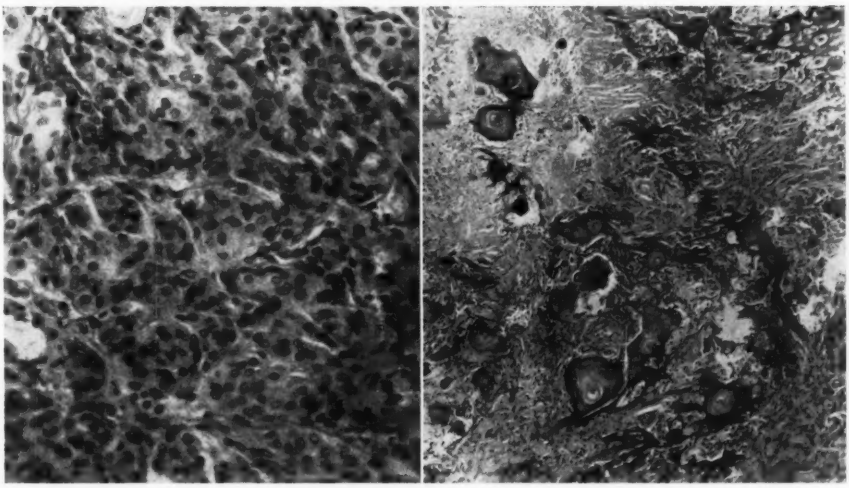

FIG. 2. (*Left*) Section of meningothelial area of the tumor. (Right) Area with psammomatous formations. (Hematoxylin-eosin, ×114 and ×57)

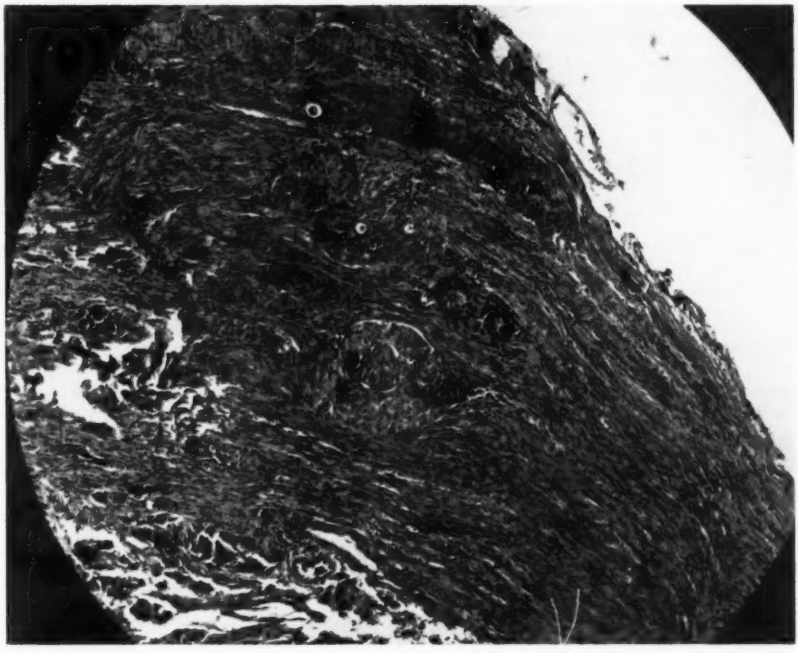

FIG. 3. Nest of tumor cells infiltrating fibers of 7th and 8th cranial nerves. (Hematoxylin-eosin, ×90)

the operation became total after it. There were very slight cerebellar signs and the patient left the hospital on the 8th postoperative day with no other neurological deficits than those related to the facial and auditory nerves.

Histological examination by Dr. Ricardo Fusté revealed a meningioma with psammomatous formations (Fig. 2). In other sections it can be seen how the tumor cells appeared infiltrating the 7th and 8th cranial nerves (Fig. 3).

Subsequent Course. Anastomosis of the left hypoglossal to the left facial nerve was done Aug. 3, 1955, under general anesthesia, with no technical difficulties. Postoperative course was uncomplicated.

On Oct. 6, 1955, when the patient was seen again, there was still no evidence of reinnervation of the facial muscles. At that time a keloid formation had developed on the scar of the suboccipital incision and another was beginning to develop on the scar of the anastomosis.

She was last seen on Jan. 26, 1956. There was evidence of good reinnervation of the left facial muscles, particularly those of the lower part of the face.

COMMENT

Because of the neurological signs and the changes seen in the radiograms, we felt quite sure that we were dealing with a space-occupying lesion in the region of the left cerebellopontine angle, but were at a loss regarding the histopathological type of the lesion. A number of possibilities such as an epidermoid cyst, an acoustic neurinoma, a meningioma, or a sarcoma were considered, but we had no real basis to favor any of them until the lesion was surgically exposed. The histological picture corresponded to that of a typical meningioma.

We believe that we accomplished a total removal of the lesion, although we are aware that this cannot be conclusively stated at this time since there is a possibility of microscopic invasion of the petrous bone from which the tumor could grow out again.

SUMMARY

A case of a 3-year-old child with a meningioma of the internal auditory meatus which was completely removed is described. This is, to our knowledge, the first patient under 15 years of age with a meningioma of the internal auditory meatus reported in the medical literature.

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AN APPARATUS FOR PHOTOGRAPHY OF TRANSILLUMINATION OF THE HEAD

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(Received for publication October 28, 1955)

The transmission of light through the head has long been recognized as a diagnostic aid for hydranencephaly. A flashlight is the most common instrument used for such an examination. A technique for transilluminating and photographing the head has been devised to produce a permanent record of this examination. A similar technique has been mentioned in a publication of Hamby *et al.*[1]

The apparatus used for transillumination consists of a 16×36 in.-table with a round opening of 3 in. in diameter centered 8 in. from one end. The table top is covered with a 2-in. thick pad of foam rubber with an opening similar to that in the table. The patient is placed on the table with his head over the opening which is illuminated by a light source from below. The foam rubber pad is used to prevent the escape of light around the head as well as for the comfort of the patient. A $\frac{1}{4}$-in. sheet of Plexiglass is attached to the under surface of the table, covering the hole, to protect the patient from the light source. The light source, concealed in a light-proof box below the table, consists of a 100-watt bulb for observation and orientation, replaced by a No. 3 flash bulb for photography. A 6-in. reflector is used to hold these bulbs. The reflector is attached to a door in the light box to facilitate changing of bulbs. A cylindrically shaped reflector attached to the under surface of the table directs the light through the opening (Figs. 1 and 2, shown on page 220.)

Photographs may be made in either black and white or color. The camera is placed above the patient at a distance that will show the head and shoulders on the photograph (Fig. 2). For black and white photographs the picture is made with normal room lighting and the No. 3 flash bulb synchronized with a shutter opening of 1/10 sec. at *f*/4.5, using Kodak Royal Pan film. For color photography, Ektachrome type B film is used with the No. 3 flash bulb in total darkness. Shutter and diaphragm openings are the same as for black and white photographs.

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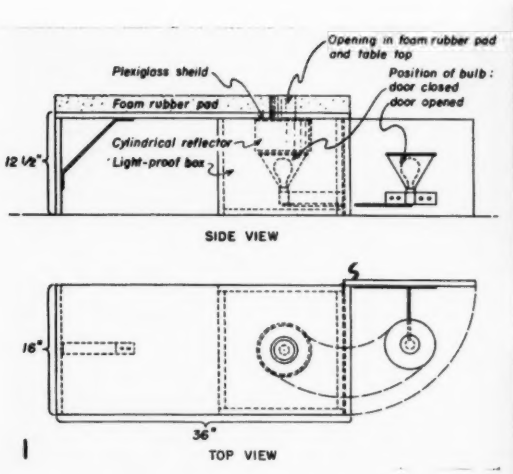

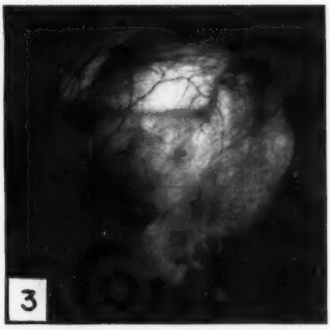

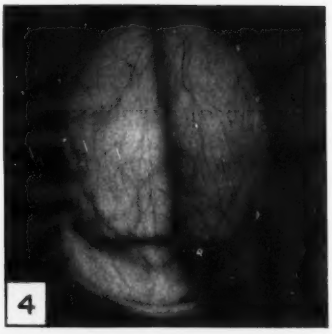

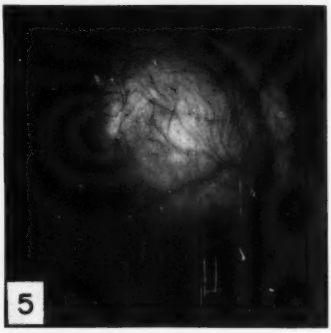

FIGS. 1–5. (1) Diagram of apparatus for transillumination of the head. (2) Set-up for making photographs of the transilluminated head. (3, 4, 5) Photographs of transillumination of the head of a 3-month-old infant. Diagnosis: Hydranencephaly.

NOTICES

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY AWARD

The American Academy of Neurological Surgery announces its second annual award. This award is intended as an incentive to research by younger neurosurgeons. It is given for the best paper submitted reporting original clinical or laboratory research pertaining to the nervous system for which the author has had the principal responsibility. Eligibility is confined to doctors of medicine currently in training in neurological surgery or who have completed their training since June 30, 1954. Those who have been certified by the American Board of Neurological Surgery, or are Fellows of the Royal College of Surgeons, Canada, subspecialty neurosurgery, or have other equivalent certification are not eligible. The paper shall not have been accepted for publication in any journal, but may have been submitted as a thesis for a degree. When subsequently published, it shall carry the footnote, "American Academy of Neurological Surgery Award, 1956."

The recipient of the award will be expected to present the paper at the annual meeting of the American Academy of Neurological Surgery in Phoenix, Arizona, November 7–11, 1956. The expenses of the recipient, while attending the meeting and in travel from his home (or if living outside the North American Continent, from the port of entry into the United States or Canada) will be borne by the Academy. In addition, there will be an honorarium of one hundred dollars.

Manuscripts for consideration by the committee should be sent to Dr. Edwin B. Boldrey, Chairman of the Award Committee, University of California School of Medicine, San Francisco, California, and should be submitted before July 15, 1956. A résumé of the candidate's postdoctoral training must accompany the paper when submitted.

AMERICAN ELECTROENCEPHALOGRAPHIC SOCIETY

The 10th annual meeting of the American Electroencephalographic Society will take place at the Claridge Hotel, Atlantic City, New Jersey on June 15, 16 and 17, 1956 immediately preceding the annual meeting of the American Neurological Association. The scientific sessions will be held Friday afternoon and evening, all day Saturday, and Sunday morning.

The program includes a colloquium on "The History of Neurophysiology" Friday evening, June 15, under the Chairmanship of Dr. Alexander Forbes. A symposium on "EEG in the Diagnosis of Coma States" will be held Sunday morning, June 17, under the Chairmanship of Dr. Jerome K. Merlis.

The annual banquet will take place Saturday evening, June 16.

SOCIETY OF NEUROLOGICAL SURGEONS

The next meeting of the Society of Neurological Surgeons will be held in Memphis on May 7 and 8, 1956. Hotel headquarters will be the Peabody. Attendance is for the members and invited guests.

JOURNAL OF NEUROSURGERY

The *Journal of Neurosurgery* is devoted to the publication of work relating primarily to neurosurgery, including studies in clinical neurophysiology, organic neurology, ophthalmology, radiology and pathology. Short articles on unusual cases and on technique, special instruments or equipment that might be useful to others in the field are also acceptable.

MANUSCRIPTS should be addressed to Dr. Louise Eisenhardt, 333 Cedar Street, New Haven 11, Conn., U.S.A. They should be typewritten in double space, with tabular matter and passages for small print on separate sheets. All materials should be mailed flat.

REFERENCES should be arranged alphabetically and cited by superior numerals in the text. The following order should be used:

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ILLUSTRATIONS. Care should be taken not to use too many illustrations, since the Journal provides no figures free of charge. Color plates also are inserted at the expense of the contributor. If illustrations are larger than 8.5×11 inches, reduced glossy prints should be submitted instead.

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